# Effects of self-regulated recovery during high intensity training on performance adaptations

A thesis submitted for the degree of Doctor of Philosophy (PhD)

by

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December, 2017

## Declaration

Candidate's declarations:

I, Oliver James hereby certify that this thesis submitted in partial fulfilment of the requirements for the award of Doctor of Philosophy (PhD), Abertay University, is wholly my own work unless otherwise referenced or acknowledged. This work has not been submitted for any other qualification at any other academic institution.

Signed [candidates signature]…..…….
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## Certificate of Approval

I certify that this is a true and accurate version of the thesis approved by the examiners, and that all relevant ordinance regulations have been fulfilled.

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## Acknowledgements

Thank you to my supervisory team Dr John Babraj and Dr Luis Calmeiro, without your continuous support and guidance I would never have completed this thesis, nor would I have developed the skills and knowledge I now possess.

Thank you to my undergraduate and Masters supervisor Dr Shaun Phillips, your help during my dissertation not only motivated me to begin my post graduate research but also gave me the starting block for my thesis topic.

Thank you to each participant that took part in my research, without your help it would not have been possible for me to complete this thesis.

Thank you to the laboratory technician Scott Marshall for working early and later then required to ensure that all the necessary lab equipment was working.

Finally, I would like to thank the continuous support of my family, friends, hockey club (GMHC), and my partner Meghann. You have all helped me keep my sanity and happiness during the three years it took me to write this thesis.

## Abstract

The overall aim to this thesis was to identify if the use of self-regulated (SR) rest during high intensity training (HIT) can be used to increase endurance and power output measures between males and females.

Study one aimed to determine if males and females can maintain mean power output (MPO) during repeated sprints when using self-regulated (SR) rest, and identify male and female response in MPO when SR rest is reduced. Participants completed four trials of 10 x 6 sec sprints separated by SR rest against 7.5% body mass (BM) as a resistance. If the mean power output (MPO) for all ten sprints in each trial had a coefficient of variation (CV) of ≤ 5.2%, then it was deemed that the participant was able to maintain their MPO. In trials 1-4 males significantly maintained their MPO greater than females in relation to their respected criterion sprint MPO data. In addition to this, only 85% of the participants could maintain their MPO when using SR rest (two males and one female failed). When SR rest was reduced by 10 and 15% there was no difference in CV between these two trials and the original 4 trials. However, MPO significantly dropped greater in females than in males SR rest was reduced by 15%. Therefore, this study indicates that males can use SR rest to maintain their MPO greater than females, and participants may be pacing their sprint efforts to maintain a sub-maximal MPO instead of their maximal MPO when SR rest is reduced by 15%.

Study two aimed to compare endurance and Wingate power output adaptations to HIT with a fixed rest (30 sec) or self-regulated rest, and identify if reproducibility of MPO during HIT is correlated to endurance and Wingate power output adaptation. Male participants the same HIT protocol from Study 1 for six sessions over a two-week period. Participants completed the HIT with either SR rest or with a fixed rest (FR) of 30 sec between each sprint. Magnitude in change for time to exhaustion (TTE), time trial (TT) and Wingate power measures was greater in the SR group, whereas $VO_2$ peak increased greater in the FR

group. However, no strong correlation between maintaining power output and increasing endurance measures or power measures appeared. Whereas correlation data indicates that $VO_2$ peak increased for the FR group due to a decrease in power output during the trials. Therefore, this study indicates that TTE, TT and Wingate power output experience a greater increase when rest is SR and with the aim of maintaining MPO during HIT.

Study three aimed to compare the magnitude in change in $VO_2$ peak, TTE, TT, and critical power (CP) when SR rest is reduced by 15 and 20% during HIT between males and females. Participants completed the same HIT protocol from the previous studies but completed eight HIT sessions over a four week period. Both training groups experienced a significant increase in endurance performance as measured via $VO_2$ peak (males only), TTE, TT and critical power (20% group only). A larger aerobic response during the HIT was significantly correlated to an increase in $VO_2$ peak in both males and females. Increases in critical power was significantly correlated to an improved TT time, which was also significantly correlated to increasing TTE. Indicating that TTE and TT improved due to an increase in greater power output. Reducing SR rest leads to a greater increase in endurance measures compared to non-reduced SR rest (Study 2), apart from females $VO_2$ peak who saw no change.

*Conclusion:*

Overall this thesis can conclude that: 1) males appear to maintain their MPO greater than females when using SR rest and females experience a greater drop in trial MPO when SR rest is reduced. 2) Participants may be pacing in trials as CV remains unchanged but MPO decreases. 3) The CV method to identify successful SR rest to maintain MPO is unreliable and doesn't take into account potential pacing tactics. 4). SR rest in HIT causes a greater increase in TTE, TT and Wingate power output measures, however, using a FR leads to greater increases in $VO_2$ peak all in males. 5) Reduced SR rest causes a greater increase in TTE, TT

and $VO_2$ peak (males only) compared to SR rest, and also increases CP. 6) Maintaining MPO during HIT is not strongly correlated to increasing endurance or power output measures.

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# 1 General Introduction – Chapter 1

## 1.1 Energy systems

### 1.1.1 Adenylate kinase

During intense bouts of exercise adenosine triphosphate (ATP) can be resynthesized through the enzyme adenylate kinase when ATP cannot be resynthesized through phosphocreatine (PCr), glycolysis or aerobic metabolism (Glaister., 2005). This process involves paring adenosine diphosphate (ADP) to create ATP and adenosine monophosphate (Equation 1.1 (Glaister., 2005)).

$$ADP + ADP \leftarrow^{\text{adenylate kinase}} \rightarrow ATP + AMP$$

*Equation 1. 1: ATP turnover through adenylate kinase activity. AMP, adenosine monophosphate.*

Adenosine monophosphate (AMP) is further deaminated to create inosine monophosphate (IMP) and ammonia through the reaction of the enzyme AMP deaminase (Equation 1.2 (Glaister., 2005)).

$$AMP + H+ \leftarrow^{\text{AMP deaminase}} \rightarrow IMP + NH4+$$

*Equation 1. 2: Role of AMP deaminase to producing AMP and IMP. Where NH4+ is ammonia and H+ is hydrogen ion.*

### 1.1.2 Adenosine Triphosphate

In order for muscular contraction, regardless of intensity, the human body obtains energy from ATP hydrolysis from the enzyme ATPase (Equation 1.3).

$$ATP \xrightarrow{\text{ATPase}} ADP + Pi + \text{energy}$$

*Equation 1. 3: Reduction of ATP to produce energy. Where ADP is adenosine diphosphate, and Pi is inorganic phosphate.*

Approximately 20-25 mmol/kg dry muscle ATP is stored within the human body, during maximal activity peak ATP turnover is ~ 15 mmol/kg dry muscle per second (Gaitanos et al., 1993; Bogdanis et al., 1998; Parolin et al., 1999). This is a small amount that is only enough to fuel 1-2sec of maximal activity (Gaitanos et al., 1993; Parolin et al., 1999). In order to maintain maximal activity the human body uses multiple metabolic processes (Phosphocreatine, anaerobic glycolysis, and aerobic metabolism) to resynthesize depleted ATP.

### 1.1.3 **Phosphocreatine**

During high intensity bouts of exercise, phosphocreatine (PCr) and adenosine diphosphate (ADP) are important for the resynthesis of ATP (Glaister., 2005). PCr converts ADP back to ATP by using it's phosphate through the enzyme creatine kinase to dephosphorylate PCr into creatine (Cr (Equation 1.4)).

$$PCr + ADP + H^{+} \xleftarrow{\text{Creatine Kinase}} ATP + Cr$$

*Equation 1. 4: Using PCr for ATP turnover.*

Converting ADP and PCr into ATP is a rapid process which is short lived due to the small amounts of PCr stored within skeletal muscle cells (80 mmol/kg dry muscle (29, 32-34)). PCr stores are reduced rapidly during intense bouts of exercise after 10sec (9 mmol/kg dry muscle/sec (Hultman & Sjöholm., 1983)).

### 1.1.4 Anaerobic glycolysis

Anaerobic glycolysis is the breakdown of an immediate reserve of carbohydrates, which is stored as muscle glycogen and is used for ATP turnover. It is predominately used within slightly less intense bouts of exercise, as anaerobic glycolysis involves more reactions/ enzyme steps for ATP regeneration than breakdown of stored ATP or PCr (Equation 1.3, 1.4 and Figure 1.1). These enzyme steps include phosphorylase, responsible for the breakdown of muscle glycogen to glucose 1-phosphate, phosphofructokinase (PFK), responsible for the phosphorylation of the glycolytic intermediate fructose 6-phosphate, and lactate dehydrogenase (LDH), responsible for the

conversion of pyruvate to lactate (MacLaren & Morton., 2012). Anaerobic glycolysis is derived from the breakdown of glucose in the form of muscle glycogen, which then produces ATP and lactate (Glaister., 2005). After ~5sec of maximal intense exercise ATP turnover from anaerobic glycolysis peaks at ~6-9 mmol/kg dry muscle/sec (Hultman & Sjöholm., 1983; Jones et al., 1985).

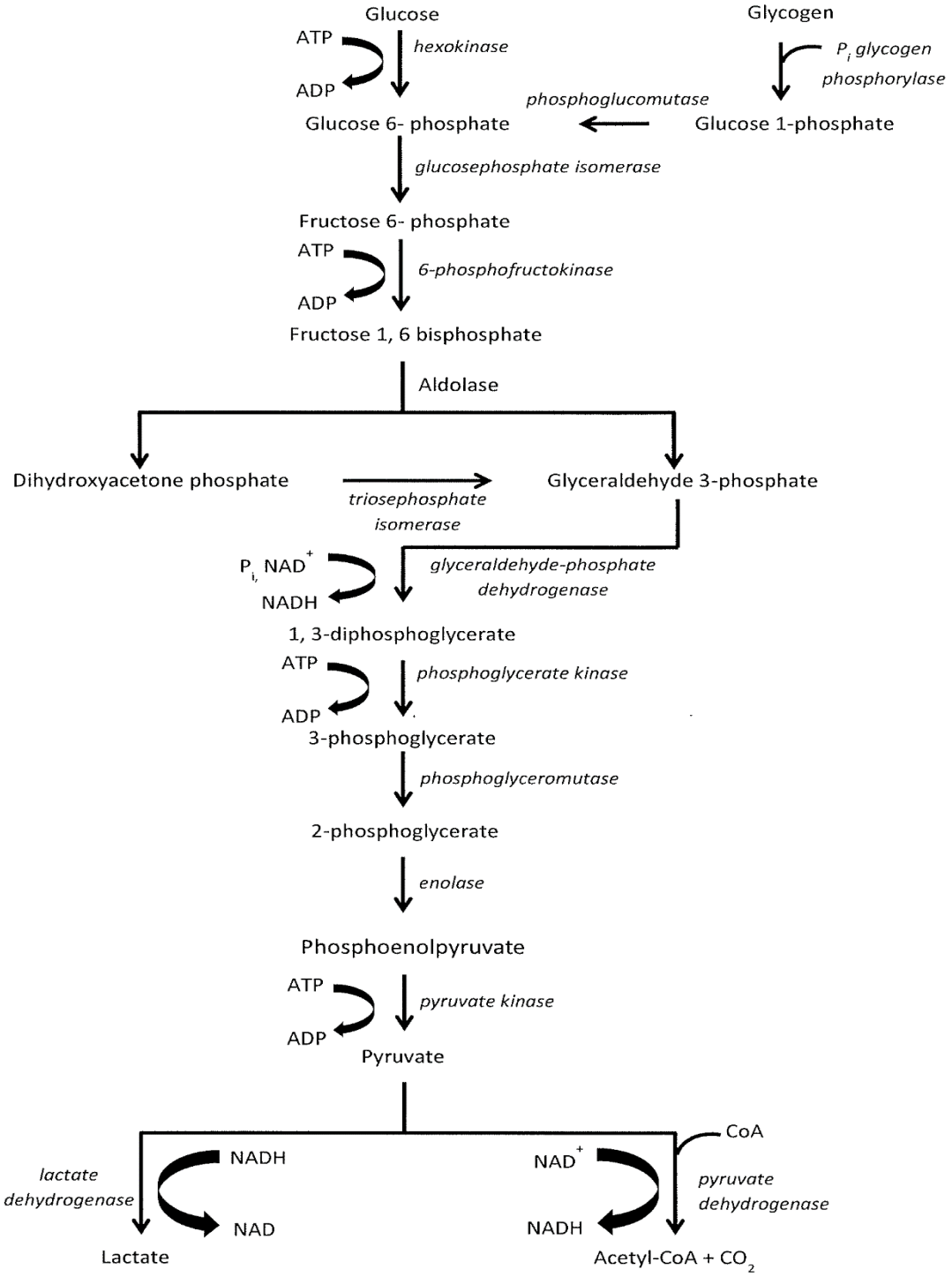


*Figure 1. 1: Anaerobic glycolysis pathway, where $NAD^+$ is nicotinamide adenine dinucleotide (oxidised), NADH is nicotinamide adenine dinucleotide, CoA is acetoacetate, Acetyl-CoA is Acetyl coenzyme A, and $CO_2$ is carbon dioxide (Maughan & Gleeson., 2004).*

### 1.1.5 Aerobic metabolism

Aerobic metabolism or oxidative phosphorylation indicates that ATP turnover comes from the breakdown of blood glucose or stored carbohydrates (glycogen) and fats (lipids) through oxidative processes within the mitochondria (MacLaren & Morton., 2012). Pyruvate, which is formed at the end of glycolysis, is converted into Acetyl coenzyme A (acetyl-CoA) and passes onto the tricarboxylic acid (TCA) cycle (Figure 1.2 (MacLaren & Morton., 2012)). Due to the use of an aerobic process, unlike PCr and glycolysis (anaerobic), there are multiple enzyme processes to produce ATP, which leads to a decrease in power output (Bogdanis et al., 1996; Gaitanos et al., 1993). Aerobic metabolism is more dominant in ATP turnover during lower intensity excise (walking, jogging (Hargreaves & Spriet., 2006)) or when a maximal sprint bout exceeds 15sec, due to a depletion in PCr and inhibited use of glycolysis (Parolin et al., 1999). Even during the first 6sec of a 30sec sprint, aerobic metabolism is responsible for ~1.32 mmol/kg dry muscle/sec ATP turnover (Parolin et al., 1999). As a 30sec sprint continues aerobic metabolism increases its rate of ATP turnover but not to the same rate as PCr or glycolysis, resulting in a slower and or less powerful sprint (Parolin et al., 1999). During multiple 30sec sprint bouts aerobic metabolism responsible for ATP turnover will increase from 29-43% from sprint one to sprint three (Bogdanis et al., 1996).

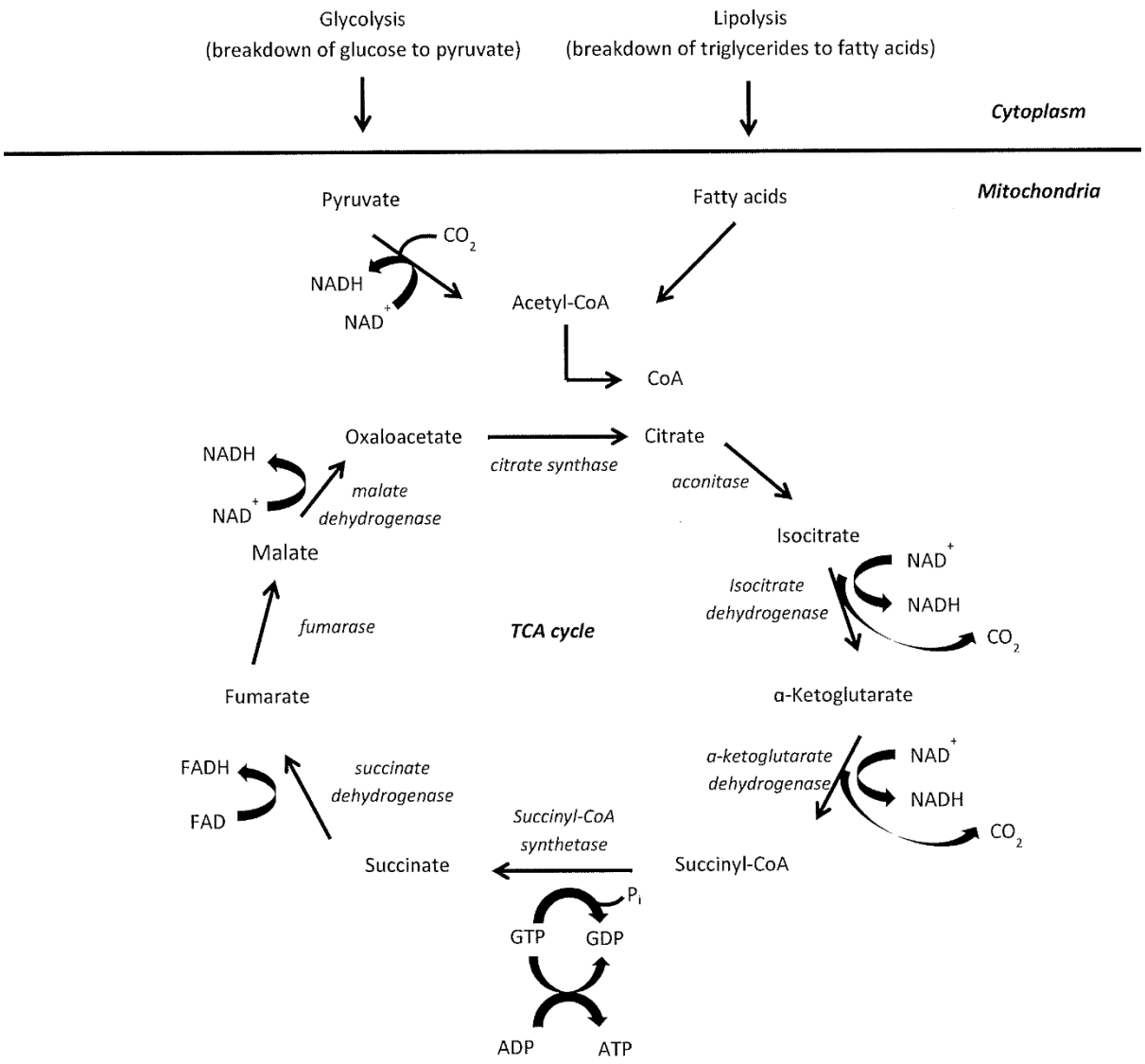


*Figure 1. 2: Aerobic metabolism pathway, where GDP is guanosine diphosphate, GTP is guanosine triphosphate, FAD is flavin adenine dinucleotide, and FADH is flavin adenine dinucleotide hydroquinone (Powers & Howley., 2009).*

## 1.2 Energy consumption during a 30 second sprint

Figure 1.3 and 1.4 demonstrates ATP turnover rate during a 30sec sprint. Bogdanis et al., (1996) identified that the calculated ATP turnover rate during two 30sec sprints separated by 4min rest. PCr + ATP are responsible for 23% during sprint one which then decreases to 20% in sprint 2 (Bogdanis et al., 1996). Similarly Glycolysis decreases from 48-36%, with oxidative

phosphorylation increasing from 29-43% during the two sprint bouts Bogdanis et al., (1996).

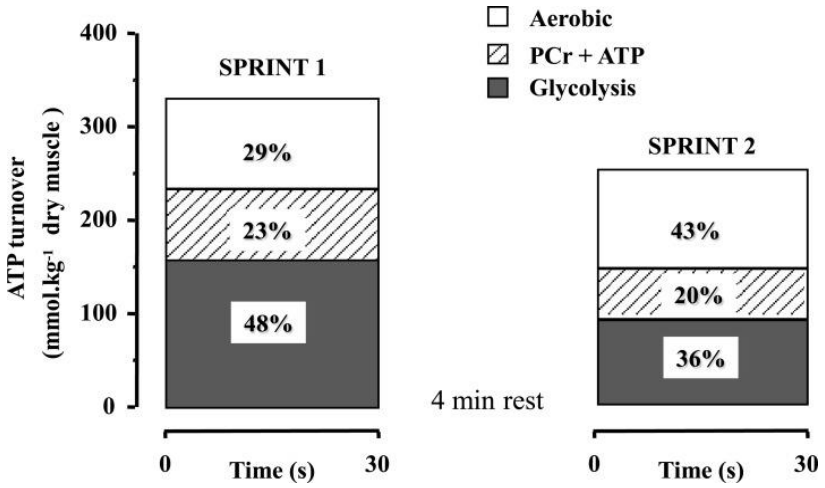


*Figure 1. 3: Calculated ATP turnover rate (%) from PCr + ATP, glycolysis, and oxidative phosphorylation during two 30sec sprints separated by 4min rest. From Bodganis et al., (1996).*

Parolin et al., (1999) conducted similar research to Bogdanis et al., (1996) but identified what is responsible for ATP turnover during 0-6sec, 6-15sec and 15-30sec sprint periods.

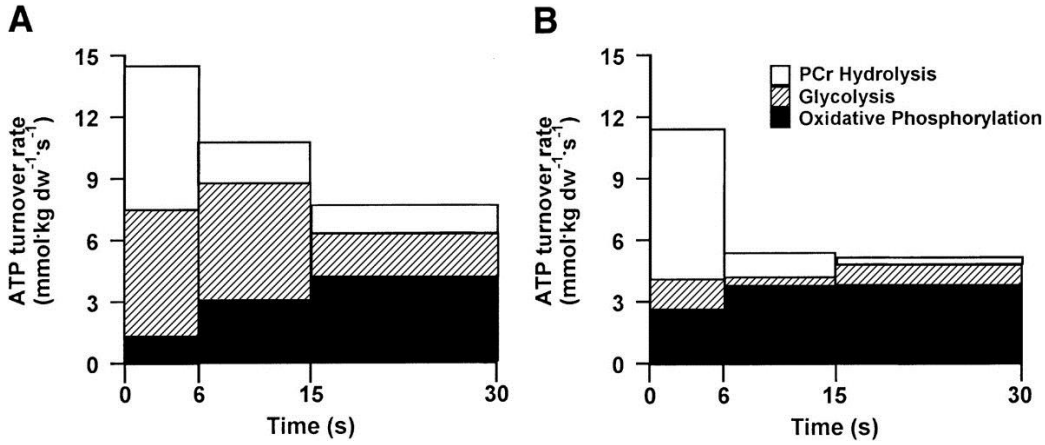


*Figure 1. 4: Calculated ATP turnover rate (mmol·kg dry $wt^{-1}$·$s^{-1}$) from PCr hydrolysis, glycolysis, and oxidative phosphorylation during bout 1 (A) and bout 3 (B). From Parolin et al., (1999).*

During the first 6sec of a 30sec sprint ATP is derived from phosphorylation by rapid PCr hydrolysis of approximately 48%, phosphorylation of glycolysis

contributes to ~43% ATP turnover, and oxidative phosphorylation is responsible for ~9% ATP turnover (Parolin et al., 1999). During 6-15sec of the 30sec sprint, power output starts to drop (Figure 1.5) due to the high demand in ATP turnover and PCr hydrolysis there is a significant increase in inorganic phosphates (Pi) of ~240% from rest to 6sec. Pi continues to steadily increase from 6-15sec (~24%) and 15-30sec (~5%). This rapid increase in Pi disrupts PCr resynthesis and calcium ($Ca^{2+}$) release (skeletal muscle contraction) and absorption (skeletal muscle relaxation) within the sarcoplasmic reticulum (Glaister et al., 2005; Ørtenblad et al., 2011; Pilegaard et al., 1999; Westerblad, Allen, Lannergren., 2002).

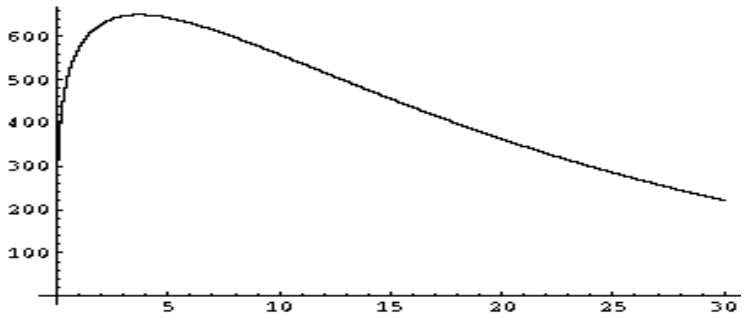


*Figure 1. 5: Typical Wingate 30sec sprint profile. Y axis represents power output (watts), and X axis presents time (sec).*

The amount of phosphorylation of PCr steadily drops to ~68%, from resting values, whereas phosphorylation of glycolysis stays elevated, with oxidative phosphorylation becoming more prominent (Parolin et al., 1999). In the last 15sec of the 30sec sprint PCr stores are depleted by ~91% from resting values, glycolysis becomes inhibited, and oxidative phosphorylation is responsible for > 50% of ATP turnover (Parolin et al., 1999).

## 1.3 Energy use during repeat sprint activity

The majority of ATP resynthesis during a 6 sec sprint is provided by PCr degradation, with a significant contribution also from anaerobic glycolysis

(Gaitanos et al., 1993; Figure 1.4). However, the metabolic contribution to ATP resynthesis during repeated 6 sec sprints is influenced by the recovery duration between sprints (Mendez-Villanueva et al., 2012). When performing 10 x 6 sec sprints against 7.5% body mass with a 30 sec passive recovery, ATP resynthesis had an increase in aerobic contribution (estimated at 13.1 mmol. kg dry $wt^{-1}$. $s^{-1}$) and a reduction in anaerobic contribution (63% reduction in glycogen contribution (Gaitanos et al., 1993)). These changes in metabolic contribution result in a decrease in both peak power output (PPO (33.4%)) and mean power output (MPO (26.6%)) between sprint one and ten (Gaitanos et al., 1993). Glycolysis becomes inhibited during repeat sprint activity however, the exact mechanism is unknown. The most plausible explanation is that glycolysis is inhibited by the progressive depletion of muscle glycogen stores that are used during this type of exercise (Balsom et al., 1999; Gaitanos et al., 1993). Unlike PCr glycogen stores can only be restored through diet (Glaister., 2005). The depletion of glycogen has been shown to impair the release of calcium ($Ca^{2+}$) within the sarcoplasmic reticulum, which decreases muscle activation and therefore force produced (Ørtenblad et al., 2011).

## 1.4 Variables that influence adaptation to HIT

### 1.4.1 Manipulating the work to rest ratio

Tables 1.1 and 1.2 show studies that have used specific work:rest ratios during high intensity training (HIT) to improve endurance and power output, with a 1:8 ratio appearing to be the most commonly used within HIT research. Work:rest ratios are designed to manipulate physiological responses to the HIT in order to stimulate desired performance improvements (Kavaliauskas, Aspe, Babraj., 2015). Performance outcomes and goals depend on training: intensity, volume, progressive overload, frequency, and appropriate methods for targeted performance outcomes (Pincivero et al., 1997). Understanding the energy pathways used during repeat sprint ability, explains why manipulating rest times can lead to specific training adaptations (Turner & Stewart., 2013). PCr is resynthesized with the use of the aerobic system, the length of rest dictates how much PCr is resynthesized with approximately 1.3 mmol/kg dry muscle per

second being resynthesised (Gaitanos et al., 1993). At least 2 minutes of passive recovery is required to synthesize at least 84% PCr normal stored levels after dynamic knee extensions at 60% maximal effort until exhaustion (Harris et al., 1976; Hultman et al., 1967). Specially in repeat sprint activity it has been found that after 10 x 6 sec cycle sprints (separated by 30 sec passive recovery against air resistance) that ~83% of PCr is resynthesized following 6 min of passive rest (Mendez-Villaneuva et al., 2012). A similar amount of PCr recovery has been found following a 2 min rest after a single 10 sec (~86%) cycle sprint against 7.5% body mass (Bogdanis et al., 1998). However, when the duration of the single sprint bout is doubled (20 sec) the amount of PCr recovery after a 2 min passive rest is decreased (~76% (Bogdanis et al., 1998)). Allowing a longer rest between sprint bouts leads to an increase in PCr resynthesis to maintain a higher rate of ATP recovery (Gaitanos et al., 1993) during subsequent high-intensity effort, enabling improved sprint speed and or power production (Billaut & Bishop., 2009). This has been found by Dawson et al., (1997), who compared PCr recovery rate following 10 sec (~55%), 30 sec (~69%) and 3 min (~90%) recovery following a single 6 sec cycle sprint against 7.5% body mass. They also found that PCr recovery was impaired following 5 x 6 sec sprints (30 sec passive recovery) compared to a single sprint at the same recovery time points of 10 sec (~27%), 30 sec (~45%) and 3 min (~84%). Given that PCr rate of recovery can be altered due to the rest duration, it may explain why using specific work:rest ratios lead to specific performance outcomes (Kavaliauskas, Aspe, Babraj., 2015). Altering rest during HIT has previously demonstrated to lead to specific performance adaptations (Kavaliauskas, Aspe, Babraj., 2015). Kavaliauskas, Aspe, Babraj., (2015) performed 6 x 10sec sprints (7.5% body mass resistance) using either a 30sec (1:3), 80sec (1:8) or 120sec (1:12) rest. They found that a shorter work:rest ratio (1:3) led to greater improvements in $VO_2$ peak (~6.9%), TTE (~6.3%) and TT (~3.1%), compared to the 1:8 group ($VO_2$ peak: ~4.7%), TTE: ~4.4%, TT: ~2.4%), and 1:12 group ($VO_2$ peak: ~0.3%), TTE: ~1.9%, TT: ~2.4%). However, using a greater work: rest ratio led to greater improvements in Wingate PPO (1:8: ~8.5%, 1:12: ~7.1%) and Wingate MPO (1:8: ~4.6.%, 1:12: ~5.3%), compared to the 1:3 group (PPO: ~4.3%, MPO: ~0.3%) Kavaliauskas, Aspe, Babraj., (2015) suggesting that 1:8 work:rest produce both endurance and power adaptations.

Kavaliauskas, Aspe, Babraj., (2015) speculate that increasing aerobic demand during recovery from HIT could be a main regulator for increasing endurance and vice versa for increasing power output. This can be achieved by manipulating the work:rest ratio (Kavaliauskas, Aspe, Babraj., 2015).

### 1.4.2 Maintenance of power during HIT:

Some HIT research has speculated at the possibility of maintaining power output leads to improvements in endurance and power output regardless of the work:rest ratio (Hazell et al., 2010; Jakeman, Adamson, Babraj., 2012; Lloyd Jones, Morris, Jakeman., 2017; Yamagishi & Babraj., 2017). Hazell et al., (2010) measured maintenance of peak power, average power and minimum power output (percentage change from sprint one) over six sessions of HIT (4-6 x 30sec sprints separated by 4min rest, 4-6 x 10sec sprints separated by 4min and 2min rest, all using 7.5% body mass resistance). They found that the 30 sec sprint group produced a significant less amount of peak power, average power and minimum power output compared to the two 10 sec sprint training groups. Hazell et al., (2010) speculates that the 10 sec with 4min rest group (1:24) improved significantly in all tests ($VO_2$ max: ~9.2%, TT: ~3.5%, PPO: ~8.5%, MPO: ~6.5%) similar to the 30sec with 4min rest group (1:8 ($VO_2$ max: ~9.3%, TT: ~5.2%, PPO: ~9.5%, MPO: ~12.1%)) due to a greater maintenance of power compared to the 1:8 group. They also speculate that 10sec sprints separated by 2min (1:12) of rest led to smaller percentage changes ($VO_2$ max: ~3.8%, TT: ~3%, PPO: ~4.2%, MPO: ~2.9%) due to a shorter rest which led to a slightly reduced mean maintenance of power compared to the 1:24 group.

Yamagishi & Babraj., (2017) compared peak and average power output reproducibility percentage between their two training groups (4-6 x 30 sec/15 sec sprints separated by 4 min/2 min rest, using 7.5% body mass for males and 6.5% for females as a resistance) in sessions 1, 6, 12 and 18. They found no significant difference between the two training groups in maintenance of PPO and MPO. With both groups significantly increasing their $VO_2$ peak (15sec: ~12.1%, 30sec: ~12.8%), TTE (15sec: ~16.2%, 30sec: ~12.8%), TT (15sec:

~8.6%, 30sec: ~7.2%) by a similar amount, and the 15sec group significantly increasing CP (15sec: ~7.8%, 30sec: ~7.4%). This further suggests that maintaining PPO and MPO during HIT could be a key factor that leads to an increase in performance adaptations, despite the reduction in HIT by 50% (Yamagishi & Babraj., 2017). However, Yamagishi & Babraj., (2017) used active recovery between sprints (40% of $VO_2$ peak) which kept cardiovascular demand high and may also have been the cause of an improvement in endurance measures (Kavaliauskas, Aspe, Babraj., 2015).

If maintaining PPO during HIT is a main regulator for performance adaptations it may explain why studies using a 6 sec sprint achieved significant improvements in TT testing, given that PPO is usually achieved within 1-5sec of a sprint (Jakeman, Adamson, Babraj., 2012; Lloyd Jones, Morris, Jakeman., 2017). Jakeman, Adamson, Babraj., (2012) found similar improvements after two weeks of HIT (10 x 6sec sprints, 1min rest, against 7.5% body mass resistance for six sessions) in TT (~10%) compared to two weeks of 30sec sprint HIT (4-6 x 30sec sprints, 4min rest, against 7.5% body mass resistance for six sessions) in TT (~9.6% (Burgomaster et al., 2006)). Lloyd Jones, Morris, Jakeman., (2017) compared 6 sec sprints against 30 sec sprints (both using 1:8 work:rest ratio against 7.5% body mass) over a two week period (six sessions) but both groups were matched for HIT duration (20 x 6sec sprints, 4 x 30sec sprints). After HIT they found a significant improvement in TT testing in both groups (6sec: ~5.1%, 30sec: ~6.2%). Both Jakeman, Adamson, Babraj., (2012) and Lloyd Jones, Morris, Jakeman., (2017) speculate that performance adaptations from HIT could be driven by the early part of a 30 sec sprint, potentially the reproducibility of PPO (Hazell et al., 2010). There is the possibility that the early part of a 30 sec sprint (0-6 sec) depletes glycogen, as long as there is multiple sprints, this depletion in glycogen could be why sprints as short as 6 sec lead to improvements in endurance performance (Jakeman, Adamson, Babraj., 2012; Knuiman, Hopman, Mensink., 2015; Lloyd Jones, Morris, Jakeman., 2017). It is thought that depleting glycogen leads to mitochondrial biogenesis which in turn would lead to an enhanced oxygen capacity within the skeletal muscle (Knuiman, Hopman, Mensink., 2015). Lloyd Jones, Morris, Jakeman., (2017) further speculates that supramaximal bouts of repeated 5-6sec sprints could be

linked with an increase in phosphofructokinase and hexokinase enzymes, along with an improved recovery period of PCr. However, none of these studies (Hazell et al., 2010; Jakeman, Adamson, Babraj., 2012; Lloyd Jones, Morris, Jakeman., 2017; Yamagishi & Babraj., 2017) actively sought to identify if maintaining peak, average and or minimum power output leads to an increase in performance adaptations.

Table 1.1: Summary of high intensity training studies on the effects of endurance capacity and performance

| Study | N | PA | Sprint Duration | Rest Duration | Study Duration | Resistance | Performance Measures |
|---|---|---|---|---|---|---|---|
| Burgomaster et al., (2005) | 6M 2F | PA | 30s | 4min | 2 weeks (6sess) | 7.5% BM | TTE↑*, & CS↑* |
| Burgomaster et al., (2006) | 8M | PA | 30s | 4min | 2 weeks (6sess) | 7.5% BM | TT↑*, & CS↑* |
| Burgomaster et al., (2008) | 5M 5F | PA | 30s | 4.5min | 6 weeks (18sess) | ~500 W | $VO_2$ peak↑*↑*, & CS↑* |
| Creer et al., (2004) | 10 | CA | 30s | 4min | 4 weeks (8sess) | 7.5% BM | $VO_2$ max↑ |
| Gibala et al., (2006) | 8M | PA | 30s | 4min | 2 weeks (6sess) | 7.5% BM | TT↑* |
| Hazell et al., (2010) | 35M 13F | PA | 30s, & 10s | 4min, & 2min | 2 weeks (6sess) | 7.5% BM | TT↑*, & $VO_2$ max↑*, ↑† |
| Jakeman et al., (2012) | 6 | CA | 6s | 1min | 2 weeks (6sess) | 7.5% BM | TT↑*, & TTE↑ |
| Kavaliauskas et al., (2015) | 10M 14F | CA | 10s | 30s, 80s, & 120s | 2 weeks (6sess) | 7.5% BM | $VO_2$ peak↑, TTE↑*,↑, & TT↑*, ↑, ↑ |
| Kavaliauskas et al., (2016) | 8F | MA | 30s | 4min | 4 weeks (8sess) | 7% BM | $VO_2$ peak→, TTE↑*, TT↑*, & CP↑* |
| Linossier et al., (1993) | 8M 2F | MA | 5s | 55s | 7 weeks (28sess) | 8% BM | $VO_2$ peak→, T1↑*, & CS↑ |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Linnosier et al., (1997) | 8M | MA | 5s | 55s | 7 weeks (28sess) | 8% BM | $VO_2$ peak↑*, & CS↑ |
| Lloyd Jones et al., (2017) | 20M | PA | 30s, & 6s | 4min, & 48s | 2 weeks (6sess) | 7.5% BM | $VO_2$ max→, & TT↑* |
| MacDougall et al., (1998) | 12M | MA | 30s | 4-2.5min | 7 weeks (21sess) | 7.5% BM | $VO_2$ max↑* |
| Perry et al., (2008) | 5M 3F | MA | 4min | 2min | 6 weeks (18sess) | ~90% $VO_2$ peak | TTE↑*, & $VO_2$ peak↑* |
| Rodas et al., (2000) | 5M | MA | 15-30s | 0.75min-12min | 2 weeks (14sess) | 7.5% BM | $VO_2$↑*, & CS↑* |
| Yamagishi & Babraj., (2017) | 10M 7F | PA | 30s, & 15s | 4min, & 2min | 9 weeks (18sess) | 7.5%, & 6.5% BM | $VO_2$ peak↑*, TTE↑*, TT↑*, & CP↑*, ↑ |

*Table 1. 1: N, number of participants; M, Male; F, Female; PA, physically active (minimum of 3-hours per week); CA, competitive athletes; MA, moderately active (< 3 hours per week); sess, sessions; BM, body mass; ↑, increase; →, no change; ↑*, significant increase; ↑†, near significant (P = 0.06); TTE, time to exhaustion; CS, citrate synthase activity; TT, time trial; VO2 max, maximal oxygen consumption; VO2 peak, peak oxygen consumption; VO2, oxygen consumption; CP, critical power; and T1, type I muscle fibres.*

Table 1.2 Summary of high intensity training studies on the effects of power output

| **Study** | **N** | **PA** | **Sprint Duration** | **Rest Duration** | **Study Duration** | **Resistance** | **Performance Measures** |
|---|---|---|---|---|---|---|---|
| Burgomaster et al., (2005) | 6M 2F | PA | 30s | 4min | 2 weeks (6sess) | 7.5% BM | TPO↑*, GLY↑*, & PCr↑ |
| Burgomaster et al., (2006) | 8M | PA | 30s | 4min | 2 weeks (6sess) | 7.5% BM | TAP↑*, GLY↑*, & PCr↑ |
| Burgomaster et al., (2008) | 5M 5F | PA | 30s | 4.5min | 6 weeks (18sess) | ~500 W | TPO↑*, TAP↑*, GLY↑*, & PCr↑* |
| Creer et al., (2004) | 10 | CA | 30s | 4min | 4 weeks (8sess) | 7.5% BM | WPO↑*, WAP↑*, |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | | | | | | RMS↑*, & MF↑* |
| Forbes et al., (2008) | 4M 3F | PA | 30s | 4min | 2 weeks (6sess) | 7.5% (M) & 6.5% (F) | TPO↑*, TAP↑*, & TPCr↑* |
| Gibala et al., (2006) | 8M | PA | 30s | 4min | 2 weeks (6sess) | 7.5% BM | GLY↑* |
| Hazell et al., (2010) | 35M 13F | PA | 30s, & 10s | 4min, & 2min | 2 weeks (6sess) | 7.5% BM | WPO↑*, & WAP↑*,→ |
| Jakeman et al., (2012) | 6 | CA | 6s | 1min | 2 weeks (6sess) | 7.5% BM | TPO↑* |
| Kavaliauskas et al., (2015) | 10M 14F | CA | 10s | 0.5min, 1.2min, & 2min | 2 weeks (6sess) | 7.5% BM | WPO↑*,↑,↑, & WAP↑*, ↑*, → |
| Kavaliauskas et al., (2016) | 8F | MA | 30s | 4min | 4 weeks (8sess) | 7% BM | TPO↑*, & TAP→ |
| Linnosier et al., (1993) | 8M 2F | MA | 5s | 55s | 7 weeks (28sess) | 8% BM | WPO↑*, WAP→, & PCr↑ |
| Linnosier et al., (1997) | 8M | MA | 5s | 55s | 7 weeks (28sess) | 8% BM | MAC↑* |
| Lloyd Jones et al., (2017) | 20M | PA | 30s, & 6s | 4min, & 48s | 2 weeks (6sess) | 7.5% BM | TPO↑* |
| McDougall et al., (1998) | 12M | MA | 30s | 4-2.5min | 7 weeks (21sess) | 7.5% BM | TPO↑* |
| Ørtenblad et al., (2000) | 9M | MA | 10s | 50s | 5 weeks (15sess) | 8-8.5%BM | TAP↑*, PCr→, GLY↑, SRv↑*, SRCr↑*, & SRCu→ |
| Perry et al., (2008) | 5M 3F | MA | 4min | 2min | 6 weeks (18sess) | ~90% $VO_2$ peak | TAP↑*, GLY↑*, & PCr↑* |
| Rodas et al., (2000) | 5M | MA | 15-30s | 0.75min-12min | 2 weeks (14sess) | 7.5% BM | WPO↑, WAP↑, GLY↑*, & PCr↑* |
| Yamagishi & Babraj., (2017) | 10M 7F | PA | 30s, & 15s | 4min, & 2min | 9 weeks (18sess) | 7.5%, & 6.5% BM | TPO↑*,↑ |

*Table 1. 2: N, number of participants; M, Male; F, Female; PA, physically active (minimum of 3-hours per week); CA, competitive athletes; MA, moderately active (< 3 hours per week); sess, sessions; BM, body mass; ↑, increase; →, no change; ↑*, significant increase; TPO, training peak power output; WPO, Wingate test peak power output; TAP, training average power output; WAP, Wingate test average power output; MAC, maximum anaerobic capacity; PCr, phosphocreatine; GLY, glycogen content; RMS, root mean square (electromyography); MF, mean frequency (electromyography); SRv, sarcoplasmic reticulum volume; SRCr, sarcoplasmic reticulum $Ca^{2+}$ release; SRCu, sarcoplasmic reticulum $Ca^{2+}$ uptake; and TPCr, reduced time take to recover phosphocreatine.*

## 1.5 Further adaptations through high intensity training

### 1.5.1 Lactate transporter activity

Anaerobic capacity and skeletal muscle power is regarded as a contributing factor for improving endurance performance (Bulbulian et al., 1986; Noakes., 1988). Increasing activity in factors such as lactate monocarboxylate transporters (MTC), specifically $MTC_1$ (associated with type I muscle fibres) and $MTC_4$ (associated with type II muscle fibres), are strongly linked with improved endurance performance, MPO and PPO performance (Pilegaard et al., 1999). Increased $MTC_{1,4}$ activity leads to an increase in skeletal muscle tissue lactate uptake, which is correlated to an increased blood flow (~16% (Gladden., 2000)). It is unclear why an increase in blood flow stimulates lactate delivery to the muscle (Gladden., 2000). However, when this does occur it promotes an intracellular shuttle in lactate from extracellular and increase lactate uptake within skeletal muscle (Gladden., 2000). As blood lactate increases 75-80% of the lactate is oxidised with the remaining 25-20% been converted into glucose and glycogen (Brooks., 2000). Therefore, allowing a greater ATP turnover to allow higher muscular contraction rates (Bogdanis et al., 1996; Gaitanos et al., 1993). Jakeman, Adamson, Babraj., (2012) used 10 x 6 sec sprints separated by 60 sec passive recovery and against 7.5% body mass resistance for two weeks (six sessions). They found that time to onset blood lactate accumulation

(OBLA) had significantly increased and speculated that this was caused by an increase in $MTC_{1,4}$ activity (Burgomaster et al., 2008). It is thought that this significant increase in time to OBLA was related to an increased work rate (30 W), which led to a ~4% increase in time to exhaustion (Jakeman, Adamson, Babraj., (2012).

### 1.5.2 Muscle metabolites and calcium dynamics

Five weeks of HIT (20 x 10sec sprint with 50sec recovery, 3 times a week, against 8-8.5% body mass) has been shown to increase $Ca^{2+}$ release (~5.5%) which is thought to be a reflection of an increase in sarcoplasmic reticulum (Ørtenblad et al., 2011). Increasing glycogen stores within skeletal muscle is also significantly correlated ($r^2 = 0.29$) to increasing $Ca^{2+}$ release (Ørtenblad et al., 2011). Increasing resting and during exercise stores of muscle glycogen (Burgomaster et al., 2005, 2006, 2008) are regulated by an increase in the glycolytic flux mechanism (MacLaren & Morton., 2012). The glycolytic flux is regulated by glucokinase activity in the liver which is a glucose sensor and increases blood glucose levels during exercise (MacLaren & Morton., 2012). Increasing phosphofructokinase and fructobisphosphatase activity increases the glycolytic shuttle and promotes insulin secretion from the pancreas (MacLaren & Morton., 2012). This in turn contributes to ~40% of the total ATP turnover during the first 15 sec of a 30 sec sprint (Parolin et all., 1999). Decreases in glycogen stores lead to an impairment of $Ca^{2+}$ release from the sarcoplasmic reticulum (Ørtenblad et al., 2011), therefore reducing the rate of muscular contraction and decreasing force production (Ørtenblad et al., 2001; Westerblad, Allen, Lannergren., 2002).

Increased PCr stores and PCr recovery has been shown after HIT (Burgomaster et al., 2006; Forbes, Slade, Meyer., 2008; Rodas et al., 2000). An Increase in PCr stores would lead to a reduction in Pi, and increase $Ca^{2+}$ dynamics (release and reabsorption rates) within the sarcoplasmic reticulum, which would lead to an increase power output (Gaitanos et al., 1993; Rodas et al., 2000; Westerblad, Allen, Lannergren., 2002). A build up in Pi and reduction in $Ca^{2+}$ sensitivity are thought to be causes for peripheral fatigue due to high

rates of PCr hydrolysis (Westerblad, Allen, Lannergren., 2002). Thus, preventing PCr resynthesis and potentially causing a decrement in power output (Westerblad, Allen, Lannergren., 2002).

### 1.5.3 Muscle fibre recruitment

HIT (4-6 30sec sprints 4min recovery against 7.5% body mass,15 x 10sec sprints with 50sec recovery against 7% body mass, 2-6 x 15 and 30sec sprints against 7.5% body mass, 4-6 x 30sec sprints 15-20min recovery against 7.5% body mass, 8-13 x 5 sec sprints against 80% optimal force), has previously demonstrated an alteration in dominance in muscle fibre recruitment during exercise post training (Allemeier et al., 1994; Esbjörnsson et al., 1993; Jacobs et al., 1987; Jansson et al., 1990; Linnosier et al., 1993). Alterations include a decrease in the recruitment of faster type muscle fibres (type IIX) and an increase the recruitment of intermediate fast twitch muscle fibres (type IIA (Allemeier et al., 1994; Esbjörnsson et al., 1993; Jacobs et al., 1987; Jansson et al., 1990; Pette., 1998; Pette & Staron., 1997; Ross & Leveritt., 2001)). HIT research has also found mixed findings with type I muscle fibre recruitment, with either a decrease (~9.4% (Jacobs et al., 1987; Jansson et al., 1990)), increase (~19%) Linnosier et al., 1993)) or no alteration (Allemeier et al., 1994). Increasing the recruitment of type I and II muscle fibres would increase the rate of glycolysis, which would increase ATP turnover, improve recovery rates through aerobic metabolism (increase PCr recovery), contribution of aerobic metabolism to power production, and greater production of force (Kent-Braun & Alexander., 2000; Pette., 1985). productions of force (Pette., 1985).

### 1.5.4 Neural adaptations

HIT training has been shown to have similar improvements in neural responses that is found within resistance strength training (Creer et al., 2004). Neural impulse responses are responsible for these alterations, due to the progression/ loading of HIT, which alters metabolic homeostasis (Pette., 1985). This results in an increased muscle fibre recruitment, firing rate, and motor unit synchronisation (Creer et al., 2004). These adaptions allow participants to exert

more force (Creer et al., 2004), which has been shown to improve 5km running time trial (~6%) by increasing running speed and running economy, despite no change in $VO_2$ max (Paavolainen et al., 1999). Neural adaptations, following 4 weeks HIT (4-10 x 30sec sprints with 4min recovery, twice a week), specifically increased motor unit activation in the vastus lateralis (Creer et al., 2004). An increase in root mean squared (~28%) and decrease in medium frequency (~17%) from surface electromyography, has been linked with increasing PPO (~6%), MPO (~6%), and lactate (~7% (Creer et al., 2004)).

## 1.6 Self-regulation

### 1.6.1 Use of self-regulation during repeat sprint activity

In repeat sprint activity (RSA) studies, participants were asked to self-select their rest periods to maintain their maximal standard of performance (Glaister et al., 2010; Phillips, Thompson, Oliver., 2014). These self-selected rest periods between sprints represent a form of self-regulation (Baumeister & Vohs., 2007; Magill., 2010; Schmidt & Wrisberg., 2007). Self-regulation is defined as individual's control of themselves seeking to perform a task with a consistent outcome, their emotions, and their instinctive behaviour (Baumeister & Vohs., 2007). It is thought that the participants in these self-regulation RSA studies used closed feedback loops to achieve an objective goal of maintaining their maximal performance (sprint speed or power output (Magill., 2010; Schmidt & Wrisberg., 2007; Weinberg & Gould., 2006)).

### 1.6.2 Closed feedback loop

Closed loop control systems are based on mechanical engineering models which can be used to describe the processes of human behaviour, if these movements require feedback then this is known as a closed feedback loop (Magill., 2010). Baumeister and Vohs. (2007) explains that feedback loops are necessary for the success of a task by using feedback. Figure 1.6 illustrates a closed feedback loop, an example of a closed feedback loop is a person

controlling their skin (shell) temperature to maintain comfort (Brooks, Fahey, Baldwin., 2004; Magill., 2010).

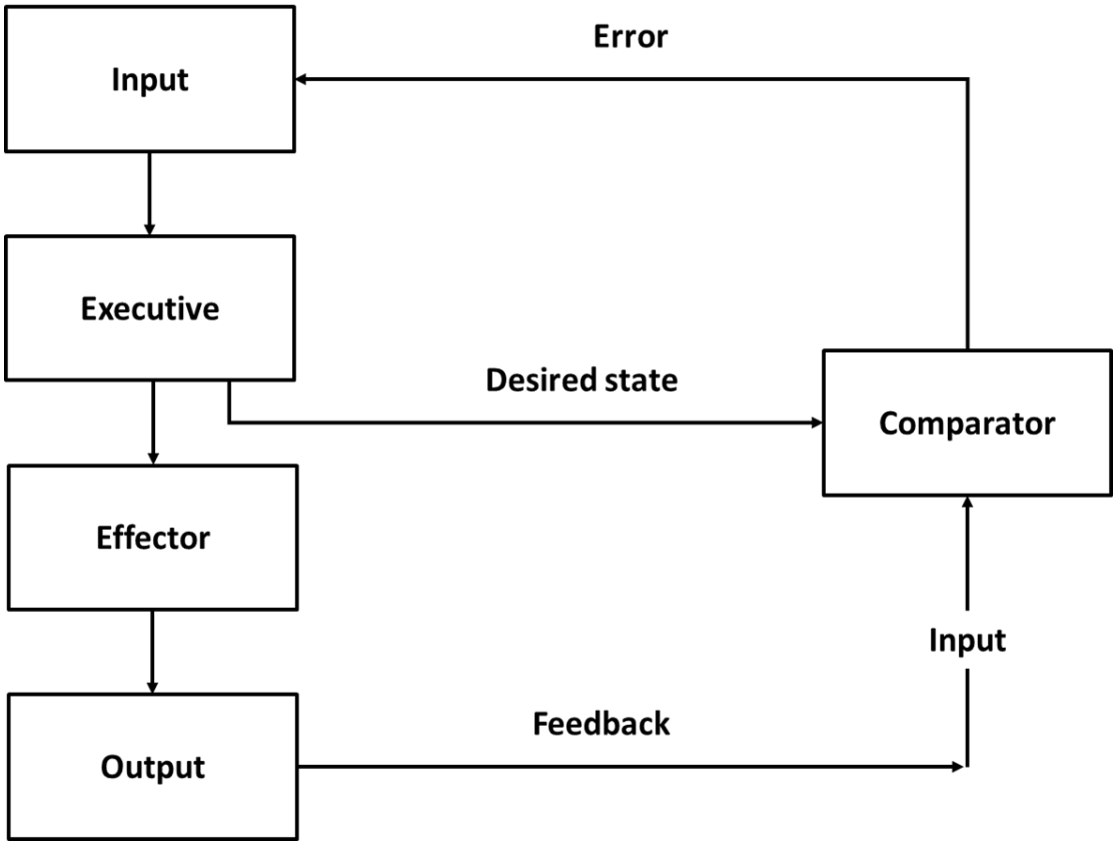


*Figure 1. 6: Schmidt & Wrisberg (2007) chart displaying the mechanisms of a closed feedback loop.*

If a person's skin temperature is too cold (Input) the person will seek to adjust their skin temperature through their own control (Executive). The person may attempt to raise their skin temperature by putting on a jacket or moving towards a source of heat (Effector), to raise the skin temperature of the body to maintain comfort (Output). The person is continuously assessing whether the skin has reached its new desired temperate (Feedback) and will identify if the changes made are appropriate in maintaining comfort (Comparator). If the changes are not appropriate (Error) then the person will repeat the process or change their approach (Input) to suit their needs of maintaining comfort (Brooks, Fahey, Baldwin., 2004; Magill., 2010). Similarly, a closed feedback loop can be in operation when participants are instructed to use self-regulated (SR) rest with the aim of repeatedly achieving their maximal running speed (12 x 30m running

sprints) or MPO (10 x 6 sec cycle sprints against 7.5% body mass resistance) (Glaister et al., 2010; Phillips, Thompson, Oliver, 2014). Participants from these SR RSA studies may have used a closed loop feedback to judge when they felt they were ready to perform their next sprint. These judgment and perception behaviours may have been controlled by peripheral sensory also known as afferent feedback (Marcora., 2008; Proske., 2005; Schmidt & Wrisberg., 2007). Afferent feedback comes from sensory nerves located within muscle spindles and Golgi tendon organs (Marcora., 2008; Proske., 2005 (see section 1.6.5 for a review)). Participants during SR RSA (with the aim of maintaining sprint speed or power output) may feel too much tension in their legs to begin their next sprint (Input). The participants may decide (Executive) to rest for a longer period of time (Effector), which may allow the feeling of tension in their legs to decrease (Output) and possibly indicate that they are ready to begin the next sprint (Feedback). After the completion of their next sprint, they will identify if the sprint they just performed equalled to what they felt was their maximal speed or power output (Comparator). The comparator may also be used when participants are comparing the tension in their legs against the lack of tension in their legs which they may associate with completing another successful sprint. After a sprint participants may then feel too much tension in their legs again to immediately begin their next sprint (Error), which then causes this closed feedback loop to occur again in order for the participants to decide when to begin the next sprint (Magill., 2010). If a sprint was successfully maintained (sprint speed or MPO maintenance) during a trial and the participant was informed of being successful, then this could be an example of the outcome response theory (Wit & Dickinson., 2009). The outcome response theory involves a goal (outcome), in this case is maintaining speed or MPO, which leads to an action (response (Wit & Dickinson., 2009)). The goal may be achieved due to a participant waiting for the tension in their legs to decrease before starting their next sprint (outcome response). A successful outcome response may lead to a greater perspective of how to maintain sprint speed or MPO through the associative-cybernetic model (ACM; Figure 1.7).

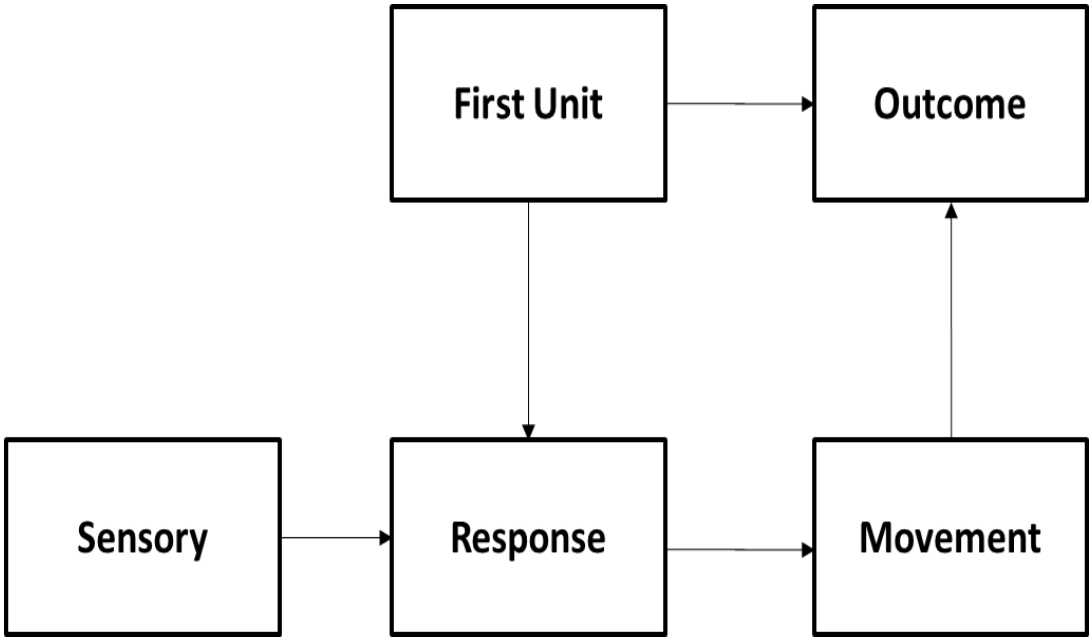


*Figure 1.7: Adapted from Wit & Dickinson (2009), chart displaying mechanisms for associative-cybernetic model.*

Specifically to SR RSA studies, possibly after a successful trial, the ACM explains the participant's readiness by identifying they are ready to begin their next sprint (first unit) with the aim of achieving a repeat of their maximal effort (outcome). The participant may feel they are ready to perform their maximal effort due to the sense of tension in their legs decreasing (sensory), which leads to the participant deciding to sprint (response) and then perform the sprint (movement). This ACM model may even explain why two familiarization trials of cycle sprints leads to a greater reliability of power output in following trials (Hopkins, Schabort, Hawley., 2001). A learning effect appears to have occurred in SR RSA studies, indicated by the reduction in PPO, MPO and sprint speed CV in the latter trials compared to the earlier trials (Glaister et al., 2010; Phillips, Thompson, Oliver., 2014). The reduction in CV would indicate a greater maintenance of power output and sprint speed (Hopkins., 2000).

### 1.6.3 Self-regulation during repeat sprint activity

Glaister et al., (2010) asked physically active, repeated-sprint habituated males (n = 20) to take part in four trials consisting of 12 x 30m running sprints. Participants were instructed to recover after each sprint for as long as they felt

was necessary in order to maintain a consistent performance across all 12 sprints. Results indicated that following two familiarisation trials, participants were able to use self-selected recovery to maintain consistent performance (within trial coefficient variation (CV) = (< 2.02%) across the 12 sprints. Despite the self-selected nature of the recovery duration and the stable sprint performance, rated of perceived exertion (RPE) increased significantly between sprints 1-12. The authors suggested that the progressive increase in RPE was an indication that participants were only just giving themselves sufficient recovery time between sprints to maintain their sprint speed repeatedly (Glaister et al 2010). However, the nature of the study protocol made elucidation of this suggestion impossible as Glaister et al., (2010) did not identify if participants were over-estimating their recovery duration. Data from Phillips, Thompson, Oliver., (2014) indicate that participants over-estimate their required SR recovery by 10% when asked to maintain their MPO. The participants saw no difference in maintaining their MPO across ten sprints with SR recovery and with a reduction of SR recovery of 10%. If the participants from Glaister et al., (2010) are also over-estimating their recovery during by 10% it would indicate that the increase in RPE data during a trial is a reflection of peripheral afferent feedback from elevated heart rate and oxygen consumption during repeated sprints (Pereira et al., 2014).

Phillips, Thompson, Oliver., (2014) recruited physically active males (n = 14) to perform four trials of 10 x 6 second cycle sprints using 7.5% body mass as a resistance and allowed participants to select their own active recovery time to mimic sprint performance across all sprints. The instructions provided to participants were identical to those reported by Glaister et al (2010). Analogous to Glasiter et al., (2010), participants were able to reliably self-regulate recovery duration in order to maintain MPO across all 10 sprints (CV = ≤ 5.2%). Participants then completed a fifth trial, where each post-sprint recovery time from participants' most reliable trial from trials 1-4 (based on within-trial lowest CV for mean power output) was reduced by 10% in a single-blind fashion. It was found that even with the reduced recovery time participants could still maintain performance across the 10 sprints; in addition, heart rate and physical rated perceived exertion (P-RPE) were also similar to those of reduced rest.

This data indicates that participants over estimate their required recovery by at least 10%. Phillips, Thompson, Oliver., (2014) also reported increased P-RPE with increasing sprint number. They speculate that the increase in P-RPE is possibly caused by a progressive increase in intramuscular acidosis and/or increased cardiorespiratory demand from the progressive increase in aerobic contribution to repeated sprints. However, P-RPE data may not be a reliable tool for suggesting an increase in intramuscular acidosis and/or increased cardiorespiratory demand. It has been previously found that P-RPE and RPE increase linearly the closer participants think they are getting to the end point of exercise (Baden et al., 2005; Swart et al., 2012). Participants will display symptoms to match their expectations in a term known as symptom belief, which has a large influence on perceived symptoms (Pohl et al., 1997). Therefore, increases in RPE and P-RPE scores in previous self-regulated studies (Glasiter et al., 2010; Phillips, Thompson, Oliver., 2014) are possibly due to the participant's knowledge or expectation of the end point of exercise. This assumption is further suggested by Baden et al., (2005), who indicates that regulation and fatigue may be a psychological response rather than a physiological response. Baden et al., (2005) had participants (male n = 8; and female n = 8) running at 75% of their peak treadmill running speed for 20 min (trial 1), 10 min then informed to run another 10 min (trial 2), and then not informing participants of running duration but still running for 20 min (trial 3). RPE measurements were significantly greater in trial 2 compared to trials 1 and 3 during minutes 11-14. Baden et al., (2005) suggests that in trial 2 at 11-14 min time points participants thought they had finished their previous task despite running at the same speed as the other two trials. Therefore, the linear increase in RPE for Phillips, Thompson, Oliver., (2014) and Glaister et al., (2010) could be due to participants having the knowledge of completing the desired amount of sprints. RPE/ P-RPE measurements during SR repeat sprint activity may not be reliable as participants may increase their RPE/ P-RPE purely because they have the knowledge that they have nearly finished the desired amount of sprints (Baden et al., 2005).

### 1.6.4 Pacing tactics

Glaister et al., (2010) and Phillips Thompson, Oliver., (2014) demonstrated that participants could use self-regulated (SR) rest periods between running sprints and cycle sprints to maintain maximal sprint performance (CV = ≤ 5.2%). Glaister et al., (2010) have suggested that participants with a lower level of aerobic capacity would choose longer rest periods, that the longer the rest period would indicate an increase in fatigue, and this could be used as a surrogate indicator for fatigue. However, Phillips, Thompson, Oliver., (2014) suggest that participants could be using pacing strategies during self-regulated (SR) rest to prevent any homeostatic disturbances or to restore homeostasis to avoid early exercise termination (Tucker et al., 2006). It has also been found that participants will pace their efforts during a single bout of exercise when comparing 5, 15, 30 and 45 sec cycle sprints against 7.5% body mass resistance (Wittekind, Micklewright, Beneke., 2011). Despite PPO being achieved typically within the first 5 sec of a sprint (Vandewalle, Pérès, Monod., 1987), a 5 sec sprint appears to produce a significant greater amount of PPO compared to a 15, 30 and 45 sec sprints (Wittekind, Micklewright, Beneke., 2011). It could be possible that when given multiple sprints participants pace their efforts similarly to the paced efforts found within a single sprint but with different durations (Wittekind, Micklewright, Beneke., 2011). These studies suggest that individuals will pace their efforts as a plan to prioritise energy expenditure in an attempt not to disturb homeostasis (Edwards & Polman., 2013). Pre planning will depend on what the nature of the task, the importance of the task and the person's capabilities and willingness to do the task (Edwards & Polman., 2013).

### 1.6.5 Peripheral or central feedback?

During self-paced cycling exercise, evidence suggests that the exercise is regulated through sensory feedback to the central nervous system (CNS) through central fatigue (Davis., 1995; Froyd et al 2016; Meeusen et al., 2006; Swart et al 2012; Kay et al 2001; Noakes, Peltonen, Rusko., 2001; St Clair Gibson et al., 2001; Swart et al 2009; Tucker et al 2006). Central fatigue is defined as a reduction in maximal capacity of the CNS to optimally recruit motor

units to produce force (Gandevia., 2001). Central fatigue is used to ensure that the participant's peripheral critical threshold is never exceeded (Amann., 2011; Amann., 2012). The peripheral critical threshold is thought to be a reduction in muscle force output due to paced efforts from an increase in peripheral fatigue in an attempt to not disturb homeostasis (Froyd et al., 2016; Hureau et al., 2014). It is also thought that peripheral fatigue is a regulator for self-paced cycling exercise by reducing the amount of muscle recruitment through afferent feedback (from peripheral organs: lungs, heart and skeletal muscle (Amann., 2011; Amann., 2012; Froyd et al 2016)). Afferent feedback comes from sensory nerves located within muscle spindles and Golgi tendon organs (Marcora., 2008; Proske., 2005). These sensory nerves sense tension, position and movement, and then send signals through the CNS to give the sense of effort (Proske., 2005). Both central and peripheral fatigue are believed to contribute to neuromuscular fatigue (Froyd et al 2016). It has been demonstrated that when intensity during exercise bouts increase there is also an increase in neuromuscular and peripheral fatigue (Amann & Dempsey., 2008). However, these studies have used self-paced time trial cycling and not looked at central or peripheral fatigue during rest periods. Therefore, the rate of peripheral and central fatigue could alter during each rest period as the body recovers during HIT. Time trial cycling and HIT may have similar rates of peripheral and central fatigue due to an increase in neuromuscular fatigue during the latter stages of their respected protocols (Froyd et al., 2016; Mendez-Villanueva et al., 2012). What can be highlighted from these studies is that peripheral fatigue occurs 20% into the time trial and steadily increases as the time trial continues (Froyd, Millet, Noakes., 2013). Whereas central fatigue is thought to occur when after peripheral fatigue had already developed (Decorte et al., 2012) and only further develops depending on the exercise duration (Place et al., 2010). Despite the lack of research in central and peripheral fatigue during HIT specifically in recovery periods, it could be suggested that peripheral fatigue will play a larger role the exercise and may therefore control SR rest. That is to say that SR rest during repeat sprint activity could be mainly controlled by afferent feedback.

## 1.7 Sex differences

While males are typically stronger and more powerful than females there is evidence indicating that females have the ability to resist peripheral fatigue to a greater extent than males (Billaut & Bishop., 2012; Hunter., 2014; Laurent et al., 2010; Perez Gomez et al., 2008; Roepstorff et al., 2006; Russ et al., 2005; Smith & Billaut., 2012). It is thought that this is due to differences in muscle mass (high in males) and fat mass (high in females), muscle fibre type distribution and recruitment, and metabolic activity differences during exercise (Billaut & Bishop., 2012; Hunter., 2014; Laurent et al., 2010; Perez Gomez et al., 2008; Roepstorff et al., 2006; Russ et al., 2005; Smith & Billaut., 2012). During repeat sprint exercise, it has been demonstrated that males have a larger recruitment in type II muscle fibres whereas females have a larger recruitment in type I muscle fibres, with the latter been associated with producing less force (Hunter., 2014). The greater muscle mass within males has been reported to increase PPO, and a greater fat mass within females have been reported to lead to a lower PPO (Perez Gomez et al., 2008). Males also have a greater activity of glycolytic enzymes and lower oxidative capacity which is in keeping with a greater development of force and recruitment of type II fibres (Russ et al., 2005). However, the greater use of glycolytic enzyme activity is greatly associated with fatiguing metabolites, such as Pi, which may lead to a greater decrement in force for males compared to females (Billaut & Bishop., 2012; Laurent et al., 2010; Smith & Billaut., 2012).

Research from Billaut & Bishop., (2012) identifies fatigue between sexes in a sporting context by comparing results between males and females during repeat sprint exercise. The study involved 35 team sport athletes both male (n = 18) and female (n = 17) completing 20 x 5 sec cycle sprints, using 9% of their body mass as a resistance, with a 25 sec rest in between each sprint. Mechanical work ($J.kg^{-1}$) achieved in sprint one and across all 20 sprints was higher in men. However, men had a significant decrease in mechanical work when compared to women across all sprints (Males: ~35%; and females: ~24%). Smith & Billaut., (2012) investigated fatigability between sexes during repeated sprint exercise. The study consisted of males (n = 10) and females (n

= 10) taking part in 10 x 10 sec cycle sprints against a resistance of 9% body mass with 30 sec rest. When sexes were matched for initial sprint work (closely ranking the results of each participant after a single 10 sec sprint), they had similar declines in work output (Males: ~27%; and females: ~30%). Laurent et al., (2010) also researched sex differences during repeat sprint activity. The study involved male (n = 8) and female (n = 8) participants completing four trials involving three bouts of 8 x 30m running sprints. Males (~40 sec) produced significantly faster times in all sprints compared to females (~44 sec). However, females had significantly lower blood lactate levels (Males: ~8.5-12.2 mmol; and females: ~7-9 mmol), and saw less of a decline in their performance (sprint time) compared to males (Males: ~5-3%; and females: ~3.5-1.7%). This occurs due to a lower use of glycolysis in females (Hunter et al., 2014; Russ et al., 2005) and a better utilisation of fat (Hunter et al., 2014; Roepstorff et al., 2006). Females also experienced a greater cardiac strain (higher heart rate) compared to males, although data was not significant it could indicate a greater oxidative load and blood flow to the skeletal muscles (Kent-Braun & Alexander., 2000). Laurent et al., (2010) concluded that males may be able to produce more power in the form of faster times, but women seem to be less fatigable than men and may recover faster. The evidence of females experiencing a smaller decline in their repeat sprint performance (Billaut & Bishop., 2012; Laurent et al., 2010; Smith & Billaut., 2012) could be due to the greater ability to recover PCr within females compared to males (Kent-Braun & Alexander., 2000). The female ability to resist fatigue greater than males is summarised in Figure 1.8.

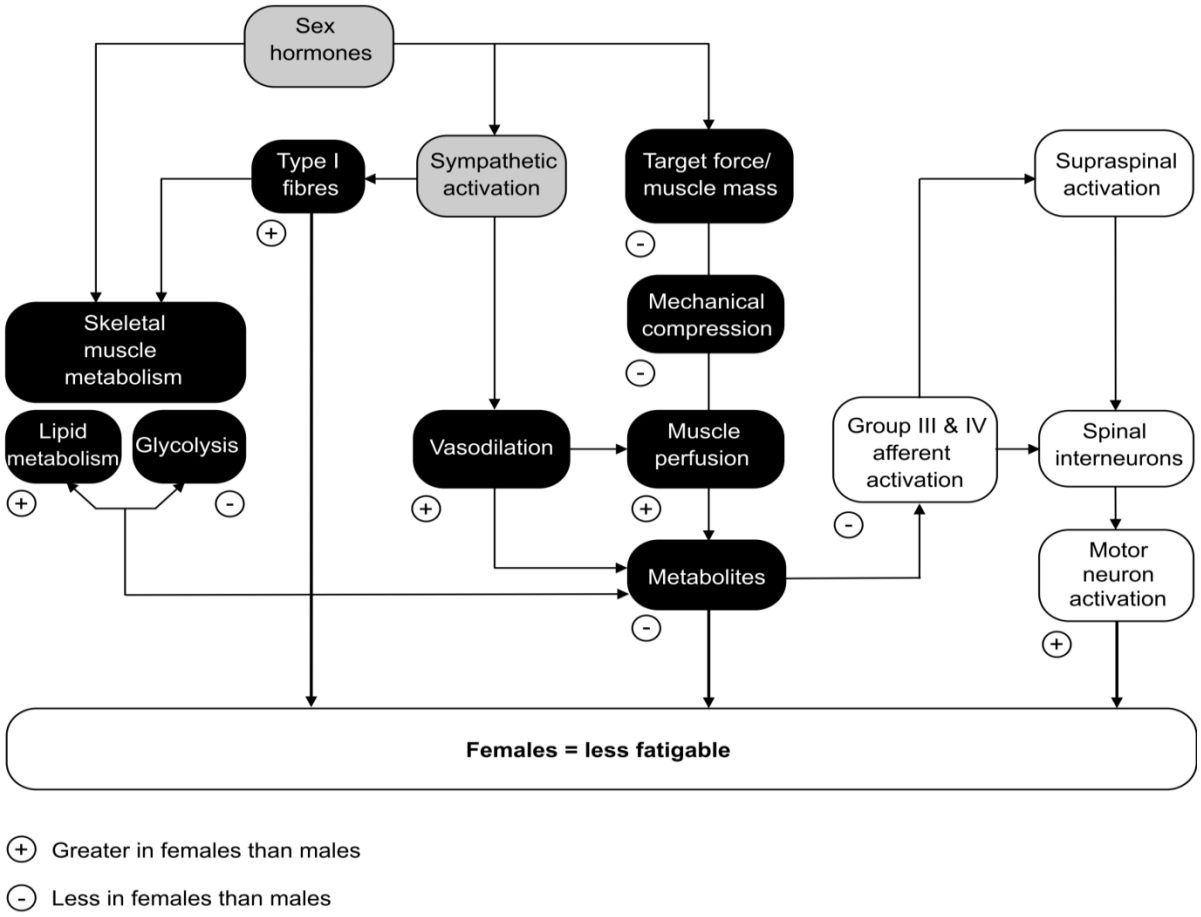


*Figure 1.8: Hunter., (2014) chart displaying potential physiological mechanisms for the sex difference in muscle fatigability, indicating that females may be less fatigable than males. Not all components from this figure are discussed in this study.*

### 1.7.1 Differences between sexes in high intensity training performance adaptations

Tables 1.1 and 1.2 indicate that endurance and power improvements can be made in both males and females following as little as two weeks of HIT. However, there is research that suggests that females do not see the same increases in $VO_2$ max that males experience following HIT (Vigelso, Andersen, Dela., 2014). Vigelso, Andersen, Dela., (2014) compared HIT and endurance training published research from 1983-2013. They found that there is a positive link between citrate synthase activity and $VO_2$ max increasing post HIT and endurance training in healthy sedentary or healthy trained young adult males.

Vigelso, Andersen, Dela., (2014) speculate that females do not have the same link in improving citrate synthase activity along with $VO_2$ max. However, the data suggesting that females do not see as strong a link in males in improved citrate synthase activity and $VO_2$ max is due to a lack of HIT/ endurance training studies that have used females (Vigelso, Andersen, Dela., 2014). Studies used within Tables 1.1 (males: n = 140; females: n = 54) and 1.2 (males: n = 234; females: n = 57) that identify the sex of their participants further indicates a lack of female presence within HIT research. There is also the possibility that the commonly prescribed 7.5% body mass resistance (see Tables 1.1 and 1.2) in the cycle sprints is too great for females due to morphological profile differences in males and females (Perez Gomez et al., 2008). With female morphological profile having a greater amount of fat mass compared to males who have a greater amount of muscle mass compared to females (Perez Gomez et al., 2008). Therefore, 7.5% body mass resistance is a greater relative intensity for females, compared to males, due to their lower muscle mass and greater fat mass (Perez Gomez et al., 2008). Hazell et al., (2010) speculates that maintaining peak power output during HIT is a key factor for improving endurance capacity and performance. Therefore, the commonly prescribed 7.5% body mass resistance (see Tables 1.1 and 1.2) might be too great to allow females to maintain their peak power output. Kavaliauskas, Steer, Babraj., (2016) saw no change in $VO_2$ peak when using only females (n = 8) and a reduced (7% body mass) resistance after 2 weeks (6 sessions) of HIT. However, there is also the possibility that female participants in Kavaliauskas, Steer, Babraj., (2016) didn't increase there $VO_2$ peak due to the short duration (2 weeks) of HIT, with longer studies (3-12 weeks) finding more participants improving their $VO_2$ max (Astorino & Schubert., 2014; Bagley et al., 2016; Gurd et al., 2016). There is also the possibility that 7% body mass resistance is too great, as Yamagishi & Babraj., (2017) found overall improvements, in both HIT groups, in $VO_2$ peak in males (n = 10) and females (n = 7) when females used 6.5% body mass resistance (Table 1.1). However, within Yamagishi & Babraj., (2017) there is also the possibility that participants increased their $VO_2$ peak due to the long duration (9 weeks) of the study (Astorino & Schubert., 2014). Astorino & Schubert., (2014) found that more sedentary participants (males n = 20, females n = 20) did not increase their $VO_2$ max (~35% of participants) when

using a short term HIT protocol (2 weeks), compared against a long term (12 weeks) HIT protocol (~22% of participants). Therefore, it is possible that female participants in Yamagishi & Babraj., (2017) improved their $VO_2$ peak due to a long duration study or due to the reduced cycling resistance.

## 1.8 Ability to self-regulate rest to maintain power and improve athletic performance between sexes

Much is to be explored about the maintenance of power output whilst using SR rest during repeat sprint cycle interval training. It is unsure if SR rest during repeat sprint training or HIT will lead to any performance adaptation (Glaister et al., 2010; Phillips, Thompson, Oliver., 2014). There is also the uncertainty if females can SR their rest to maintain performance (sprint speed and MPO). Therefore, this thesis will seek to further explore the effects of SR rest during HIT, and identify if any performance gains occur whilst using SR rest during HIT between sexes.

## 1.9 Aims

The key aims to this PhD thesis are:

To determine if males and females maintain mean power output during repeated sprints with self-regulated rest.

To identify male and female response in mean power output when self-regulated rest is reduced

Compare endurance adaptations to HIT with a fixed rest (30 sec) or self-regulated rest.

To identify if reproducibility of mean power output is correlated to endurance and Wingate power output adaptations to HIT.

To compare the magnitude in change of $VO_2$ peak, time to exhaustion, 10km time trial, and critical power between 15 and 20% reduced self-regulated rest during repeat sprint training between males and females.

# 2 Chapter 2 – Study 1 (Sex comparison during repeated cycle sprint exercise with self-regulated recovery)

## 2.1 Introduction

Whilst males are typically stronger than females there is evidence of a greater resistance to peripheral fatigue in females (Laurent et al., 2010; Smith & Billaut., 2012). This difference in the rate of peripheral fatigue has been proposed to be due to differences in percentage area of skeletal muscle fibre type between males and females (Glenmark, Hedberg, Jansson., 1992; Hicks, Kent-Braun, Ditor., 2001). Females have been found to have a significantly greater percentage area of type I muscle fibres with males showing a significantly greater percentage area of type IIa muscle fibres in the vastus lateralis (Roepstorff et al., 2006; Staron et al., 2000). The greater percentage area of type IIa muscle fibres in males is in keeping with demonstrating a higher glycolytic enzyme activity and lower oxidative capacity (Roepstorff et al., 2006; Russ et al., 2005) which leads to a greater force development (Russ et al., 2005). Females utilise glycolytic enzyme activity to a lesser extent and instead have a greater fat oxidation capacity due to a higher percentage area of type I muscle fibres compared to males (Roepstorff et al., 2006; Staron et al., 2000).

These differences may affect ability to perform sprints (Billaut & Basset., 2007; Laurent et al., 2010; Smith & Billaut., 2012), which can be regarded as an important aspect of team sport (Billaut & Basset., 2007; Gaitanos et al., 1993; Glaister et al., 2008). During both repeated sprint running and cycling bouts, males see a larger decrement in speed and power output (Billaut et al., 2011; Laurent et al., 2010). This could be due to the greater ability to recover phosphocreatine (PCr) after sprints in females compared to males (Kent-Braun & Alexander., 2000). Therefore, increasing PCr availability for the next sprint during the rest periods which will aid repeatability of sprint performance (Gaitanos et al., 1993; Rodas et al., 2000). There is evidence to indicate that females have the ability to recover PCr faster than males due to a greater ability to utilise oxygen in skeletal muscles (Kent-Braun & Alexander., 2000). Kent-Braun & Alexander., (2000) findings also suggest a strong link between a higher

$VO_2$ peak and a faster PCr recovery. In addition, at the point of exhaustion females can recover faster and produce greater amounts of absolute force compared to males (Fulco et al. 1999). This may suggest that in order to recover PCr females would require a shorter rest period following maximal exertions.

Recently, Glaister et al., (2010) and Phillips, Thompson, Oliver., (2014) demonstrated that participants could use self-regulated (SR) rest periods between running sprints (12 x 30m (Glaister et al., 2010)) and cycle sprints (10 x 6 seconds, 7.5% body mass resistance (Phillips, Thompson, Oliver., 2014)) to maintain maximal sprint performance (defined as a coefficient of variation (CV) between sprints ≤ 5.2%)). Glaister et al., (Glaister et al., 2010) have suggested that participants with a lower level of aerobic capacity would choose longer rest periods, that the longer the rest period would indicate an increase in fatigue, and this could be used as a surrogate indicator for fatigue. However, Phillips, Thompson, Oliver., (2014) suggests that participants could be using pacing strategies during self-regulated (SR) rest to prevent any homeostatic disturbances that could lead to early exercise termination (Tucker et al., 2006). Phillips, Thompson, Oliver., (2014) main findings identified that male participants over-estimated their SR rest period by at least 10%. This suggests that individuals will pace their efforts as a plan to prioritise energy expenditure in an attempt not to disturb homeostasis (Edwards & Polman., 2012). Pre planning will depend on what the task is, the importance of the task and the person's capabilities and willingness to do the task (Edwards & Polman., 2013). During maximal exercise, it is suggested that central fatigue in the central nervous system is responsible for the regulation of exercise (Davis., 1995; Kay et al., 2001; Noakes, Peltonen, Rusko., 2001; Romain et al., 2006; Swart et al., 2012; St Clair Gibson, Schabort, Noakes., 2001; Swart et al 2009; Tucker et al., 2006). Suggesting that pacing tactics are used to prevent harm to the body when exercise intensity threatens homeostatic control, and that during maximal exercise fatigue is controlled by the brain rather than physiological depletion in energy sources (Baden et al., 2005; Bassett & Howley., 2000; Billaut et al., 2011; Costill, Fink, Pollock 1976; Swart et al., 2012).

### 2.1.1 Aims and hypothesis

Given this over-regulation of SR in males (Phillips, Thompson, Oliver., 2014) and potential sex differences in sprint performance (Billaut & Bishop et al., 2011; Laurent et al., 2010) further studies are required to understand the effects of SR rest during repeat sprint exercise in males and females. The aim of this study was to investigate the reliability and accuracy of SR repeated sprint performance in males and females. It was hypothesised that females would require a shorter rest period between sprints than their male counterparts and both sexes would over-estimate recovery required by at least 10%.

## 2.2 Methods

### 2.2.1 Participants

Ten physically active males (180 ± 8 cm, 78 ± 9 kg, and 43 ± 5 $VO_{2peak}$ $ml.kg^{-1}.min^{-1}$) and females (169 ± 8 cm, 63 ± 8 kg, and 33 ± 6 $VO_{2peak}$ $ml.kg^{-1}.min^{-1}$) volunteered for this study. Before taking part, participants were given written and verbal instructions about the study prior to giving informed consent. Participants also completed a physical activity readiness questionnaire to ensure there was no known health issues that would put the participants in harm by taking part this study. Participants were asked how many hours of structured exercise they participate in per week (males = 8 ± 4h, females = 7 ± 4h), which is more than the American College of Sports Medicine and American Heart Association recommended 2.5 hours of moderate physical activity (Haskell., 2007). All participants were required to be either competitive or recreational athletes, aged 18-35 years and trained > 3 hours per week. Ethical approval was received from Abertay University ethics committee and the study was carried out in line with the declaration of Helsinki.

### 2.2.2 Procedures

#### 2.2.2.1 Baseline testing

At the beginning of the session, participants' body mass (BM, kg) and height (cm) were recorded using a digital scale (Tanita SA 165A-0950U-3) and digital

stadiometer (Seca 264), respectively. The BM measurements were required for peak oxygen uptake measurements, and to identify how much resistance would be applied during a cycle sprint.

#### 2.2.2.2 Equipment

Participants completed cycle sprints on a Monark cycle ergometer (Monark peak bike 894), mean power output (MPO) for each sprint was calculated using the Monark software (Monark Anaerobic Test Software version 2.24.2, Monark Exercise AB). MPO was calculated as the overall relative mean average watt value (watts / participant's BM). Further descriptions of how MPO was used is listed below.

#### 2.2.2.3 $VO_2$ peak

Traditionally peak and maximum oxygen uptake ($VO_2$ peak and $VO_2$ max) is defined as the highest value oxygen that can be taken up and utilized by the body during incremental exercise to exhaustion or time to exhaustion test (TTE (Bassett & Howley., 2000)). Both these measures are regarded as an indicator for capacity of cardiorespiratory function (Loftin et al., 2004), and commonly used to measure exercise capacity changes pre and post intervention testing within exercise physiology (Bassett & Howley., 2000; Stevens & Dascombe., 2015). In order to assess $VO_2$ max researchers waited for a participant's $VO_2$ to plateau (no increase in $VO_2$ despite an increase in TTE) during a TTE (Basset & Howley., (1997). However, $VO_2$ does not always plateau during a TTE and has to be repeated until a plateau occurs (Basset & Howley., 1997; Rossiter, Kowalchuk, Whipp., 2006). Taking 10-30sec averages of the highest $VO_2$ value, commonly at the end of a TTE test, is known as the $VO_2$ peak, which is likely to be a similar value to the $VO_2$ max (Rossiter, Kowalchuk, Whipp., 2006). Therefore, repeating the test until a plateau in $VO_2$ occurs is not necessary to assess capacity of cardiorespiratory function (Loftin et al., 2004; Rossiter, Kowalchuk, Whipp., 2006). Study 1 involved no intervention but a $VO_2$ peak test was conducted at the start of the study to define participants' aerobic capacity

and to enable further exploration of resting and sprinting $VO_2$ data between males and females.

Participants were fitted with a heart rate (HR) monitor (Polar) and a mask connected to an online expired gas analyser (Cortex Metamax3B). Participants then mounted the cycle ergometer (Monark peak bike) and cycled for 4 minutes at 60 W. Immediately following this warm up, power output increased by 30 $W \cdot min^{-1}$ until volitional exhaustion (Jakeman, Adamson, Babraj., 2012). Participants were instructed to maintain a cadence of 60 revolutions per minute (RPM) and were verbally encouraged throughout. The test was terminated if a participant dropped below a cadence of 60 RPM for more than 5 sec or if the participant chose to stop. $VO_2$ peak was determined as the highest 30 sec average of $VO_2$ across the test. $VO_2$ peak was determined as the highest 30 sec average of $VO_2$ across the test, which has been recommended for detecting the highest processed data point (Robergs, Dwyer, Astorino., 2010).

### 2.2.3 Experimental rational

#### 2.2.3.1 Sprint resistance

Participants cycled against 7.5% body mass in accordance to previous HIT studies (Burgomaster et al., 2005, 2006, 2008; Hazell et al., 2010; Jakeman, Adamson, Babraj., 2012; Kavaliauskas, Aspe, Babraj., 2015; Lloyd Jones, Morris, Jakeman., 2017; Yamagishi & Babraj., 2017). There is evidence that within HIT that females should adopt a lower resistance, recently Yamagishi & Babraj., (2017) and Kavaliauskas, Steer, Babraj., (2016) respectively used 6.5% and 7% body mass as a resistance for their female participants. The purpose for using a lower resistance in females is due to morphological sex differences with a greater fat mass found in females and greater muscle mass found in males (Perez-Gomez., 2008). Using a lighter resistance in females might be more suitable for producing greater power outputs (Billaut & Bishop., 2009). However, one of the aims to this thesis was to maintain MPO using SR rest and not produce greater amounts of MPO. With only males shown to maintain their MPO using SR rest (Phillips, Thompson, Oliver., 2014). Using the same

resistance of body mass for both sexes within this thesis may allow further suggestions on appropriate resistance to be used for females. This will be achieved by comparing MPO, cardiorespiratory response, and pre and post performance testing measures between sexes.

#### 2.2.3.2 Sprint duration

Traditionally within HIT research, a 30 sec sprint is used (Burgomaster et al., 2005, 2006, 2008; Hazell et al., 2010; Lloyd Jones, Morris, Jakeman., 2017; Yamagishi & Babraj., 2017). However, there is increasing research that indicates that a sprint as short as 6 sec during HIT training sees similar improvements in endurance and power post training compared to using a 30 sec sprint (Jakeman, Adamson, Babraj., 2012; Lloyd Jones, Morris, Jakeman., 2017). It has been speculated that positive performance adaptations post HIT may be driven by the early stages of the sprint and possibly explaining similar improvements in time trial testing between using a 6 and 30 sec sprint (Jakeman, Adamson, Babraj., 2012; Lloyd Jones, Morris, Jakeman., 2017). Hazell et al., (2010) also speculates that the reproducibility of power output (peak, mean, and minimum) during HIT may also be a key factor for positive performance adaptations. If the reproducibility of peak power output during HIT is a main regulator for positive performance adaptations then this may be achieved using only a 6 sec sprint (Lloyd Jones, Morris, Jakeman., 2017). Given that peak power output (PPO) is usually achieved within the first 5 sec of a sprint (Vandewalle, Pérès, Monod., 1987).

#### 2.2.3.3 Criterion sprint, fatigue index and coefficient of variation

After the completion of a $VO_2$ peak test, male and female participants completed a criterion sprint (CS) test prior to taking part in fours trials of 10 x 6 sec sprints separated by self-regulated (SR) rest and cycling against 7.5% BM as a resistance. The purpose of the CS was to provide a familiarisation session for the participants, and to allow a comparison between a single sprint MPO against an average of ten sprints MPO (Phillips, Thompson, Oliver., 2014). Phillips, Thompson, Oliver., (2014) also found that SR trials average peak

power output (PPO) was significantly less than the CS PPO, MPO between CS and SR trials followed a similar pattern but was not significant. They speculate that participants may have been pacing their efforts to complete ten maximal effort sprints instead of just one sprint despite using SR rest. Within this thesis, the CS was used to identify if participants are maintaining their maximum MPO across all ten sprints in each SR or reduced rest (RR) trial. Participants were informed before the start of their next trial if they had been maintaining their MPO (compared to their CS) as part of a feedback loop (Baumeister & Vohs., 2007; see section 1.6.2). The feedback loop was used to help participants understand if what they had done in previous trials or sessions was successful in maintaining MPO (Baumeister & Vohs., 2007). Therefore, aid participants in keeping within the aim of the study of maintaining MPO across ten sprints. To measure a drop in MPO for each participant in each trial fatigue index (FI) was used (equation 2.1).

FI = (100 x [total sprint performance / ideal sprint performance]) – 100
*Equation 2.1: Where total sprint performance = sum of MPO from all sprints, and ideal sprint performance = number of sprints x greatest MPO (Fitzsommons et al., 1993).*

The FI measurement appears to be a reliable method for identifying a decrease in performance (percentage decrement (Fitzsommons et al., 1993)). FI is strongly correlated to percentage decrement in cycling (r = 0.88) and running (r = 0.75) repeat sprint activity (Fitzsommons et al., 1993). Percentage change between the CS, average MPO, and trials 1-6 MPO to identify if participants employed pacing tactics during the trials. The CS test consisted of a single maximal effort 6 sec sprint followed by cycling against 1kg for 60 sec at 60 revolutions per minute (RPM), the MPO for the sprint was recorded, and participants then sat quietly for 5 min. This CS test was repeated for a second time and if the MPO of CS sprint two was 5% greater than CS sprint one then the CS test was repeated until the MPO of the CS failed to increase by 5% (Phillips, Thompson, Oliver., 2014). During trials 1-4, participants were informed to self-regulate (whilst cycling at 50-60 RPM against no resistance) their rest

with the aim of maintaining their MPO across all ten sprints within the trial. Maintaining power output during high intensity training (HIT) has been speculated as a potential factor for improving performance post HIT (Hazell et al., 2010; Lloyd Jones, Morris, Jakeman., 2017). Using SR rest to maintain MPO would remove factors based on recovery of PCr (Glaister., 2005) which may be different for each participant possibly based off their aerobic fitness (Glaister et al., 2010). SR rest may allow participants to maintain their MPO rather than using a fixed work:rest ratio. However, there is uncertainty whether SR rest during HIT would lead to any performance adaptation due to an overestimation in SR rest by at least 10% (Phillips, Thompson, Oliver., 2014). Therefore, identifying how long participants overestimate their SR rest by and removing this overestimation could perhaps lead to positive performance adaptations. Participants were instructed to rest until the point they felt they could reproduce the same MPO or greater MPO of their CS. Participants were blind to any timing apparatus to ensure SR rest duration was based off their personal response to the task (Glaister et al., 2010; Phillips, Thompson, Oliver., 2014). SR rest duration was defined as the moment a sprint ends until after a participant counts down from 3, 2, 1 to start the next sprint, which is in keeping with Phillips, Thompson, Oliver., (2014). SR rest duration was recorded in seconds using a digital stop watch performed manually by the researcher. To identify if participants were maintaining their MPO coefficient of variation (CV) was used (equation 2.2), CV had to be ≤ 5.2% to be deemed as a successful trial (Capriotti, Sherman, Lamb., 1999; Glaister et al., 2010; Phillips, Thompson, Oliver., 2014). CV of ≤ 5.2% was chosen given that the participants were unfamiliar with this method of training Capriotti, Sherman, Lamb., (1999). If participants were familiar with this method of training or had at least six familiarisation trials, it would be expected that MPO CV could be as little as 2.5-3.3% (Capriotti, Sherman, Lamb., 1999). Therefore, given that participants were unfamiliar with this method of training and did not receive six familiarisation trials a CV of ≤ 5.2% was deemed an appropriate marker for maintenance of MPO during SR and RR trials (Capriotti, Sherman, Lamb., 1999).

CV = (standard deviation of MPO from 10 sprints / average MPO from 10 sprints) * 100

*Equation 2.2: Coefficient of variation formula.*

CV was used to compare reliability during trials in previous research (Glaister et al., 2010; Phillips, Thompson, Oliver., 2014). CV is a commonly used method for identifying of typical error that can be used for individual participants, reliably compare what is typically percentage error between trials, and is commonly used within physical, medical and social sciences (Bai, Wang, Wong., 2011; Hopkins., 2000). Sample size (number of participants) or in this case number of sprints performed in a trial (ten), can effect CV results (Hopkins., 2000). For example degrees of freedom can be > 25 (~5% bias) when using 25 participants and 2 trials, and could be 7 degrees of freedom (~21% bias) when using 8 participants over two trials (Hopkins., 2000). Therefore, within this thesis CV of MPO within each trial could vary if more sprints of a similar MPO were performed. Participants who were successful in two of the four trials (n = 17) progressed onto trial five where 10 x 6 sec sprints were performed again but with a 10% reduction in most successful trial mean SR rest (Phillips, Thompson, Oliver., 2014). Participants who were successful in trial five (n = 16) then moved onto trial six which was similar to trial five but involved a 15% reduction in SR rest.

#### 2.2.3.4 Self-regulation

In all three studies, the participants that had to self-regulate their rest periods were left completely in charge and would inform the researcher as to when they wanted to begin their next sprint by counting down from three. Allowing this much control for the participants is in line with the definition of self-regulation, control of one's self (Baumeister & Vohs., 2007), and follows similar commands from previous self-regulated repeat sprint activity (Glaister et al., 2010; Phillips, Thompson, Oliver., 2014). In order for participants to understand if they had successfully maintain their MPO (CV ≤ 5.2%) the researcher informed the participant in order to create a feedback loop for the participant (Baumeister & Vohs., 2007; see section 1.6.2). If participants were unsuccessful they have taken this feedback and adopted a longer rest period, which would allow a

greater resynthesis of ATP and therefore greater MPO (Glaister., 2005). However, it is also possible that participants may have taken this feedback and paced their efforts or held back to allow their MPO across all ten sprints to be similar. Pacing has been shown to occur when comparing single 5, 15, 30 and 45 sec sprints, with greater PPO been achieved in a 5 sec sprint compared to 15, 30 and 45 sec sprints (Wittekind, Micklewright, Beneke., 2011). Therefore, within this research it could be possible that using ten 6 sec sprints could lead to participants pacing their efforts in order to maintain a similar MPO within a trial.

### 2.2.4 Trial procedure

#### 2.2.4.1 Sprint trials warm-up

In all trials, participants were required to complete a warm up that consisted of 4 min cycling at 60 RPM against 1kg as resistance on a cycle ergometer (Monark peak bike). Once completed a sprint specific warm up was carried out; consisting of 3 x 3 second sprints against 7.5% body mass with an active 45s recovery, cycling at 50-60 RPM (with no resistance), between sprints. Participants then rested for 4 minutes prior to completing the sprint trials (Phillips, Thompson, Oliver., 2014).

#### 2.2.4.2 Trial 1

Participants completed a 6s all-out effort sprint against 7.5% body mass with the resistance dropping once they reached 110 RPM. Participants then cycled for 60s against 1kg resistance before dismounting the bike for 300s. The sprint was then repeated and if mean power output (MPO) was greater than in sprint 1 then this was taken as their CS performance. If MPO in the second sprint was ≥ 5% of the first MPO value, the test was repeated for a third time. The CS procedure was repeated until MPO no longer increased (Phillips, Thompson, Oliver., 2014). On completion, participants had a 15 minute seated recovery. Following the recovery, participants repeated the warm up procedure and began their first self-regulated recovery sprint trial, consisting of 10 x 6s sprints against 7.5% body mass with the resistance dropping once they reached 110

RPM. Participants wore a mask connected to the online gas analyser (Cortex metamax 3B) and a HR monitor (Polar) throughout the protocol.

#### 2.2.4.3 Trials 2-4

Participants completed the same warm up and self-regulated recovery sprint trial with a minimum of 48 hours between trials. To progress onto trials 5 and 6 successful performance was determined as (Phillips, Thompson, Oliver., 2014):

1. A within-trial CV for MPO of 5.2% or less (the upper CV for this type of exercise (Capriotti, Sherman, Lamb., 1999; Glaister., 2005)). Participants had to achieve this in three out of the four trials.

Two males and one female were unable to meet the above criteria, therefore 8 males (181 ± 9 cm, 78 ± 10 kg, and 44 ± 5 $VO_2$ peak $ml.kg^{-1}.min^{-1}$) and 9 females (170 ± 3 cm, 62 ± 6 kg, and 34 ± 6 $VO_2$ peak $ml.kg^{-1}.min^{-1)}$ proceeded to trials 5 and 6.

#### 2.2.4.4 Trials 5-6

In these trials, the same procedure sprint was used except a fixed rest time was applied. The fixed rest time was calculated by taking the mean rest time from each participant's most reliable trial, based on within-trial CV for MPO (males CV = 2.3% ± 0.7% and females CV = 2.8% ± 1.1%), and reducing it by 10% or 15%. This was done to determine the extent of over-estimation of required recovery duration. One female was unable to meet the above criteria in trial 5 and did not progress to trial 6.

### 2.2.5 Assessment of cardiorespiratory response

#### 2.2.5.1 Equipment

Participants wore a gas mask that was connected to a gas analyser (Metalyzer 3B gas analyser, Cortex, Leipzig, Germany) during trials and sessions. The gas analyser was calibrated before the use of each trial or session using the lab's air gas mixture of known oxygen ($O_2$) and carbon dioxide ($CO_2$). It was

assumed that partial air gas was 20.93% ($O_2$) and 0.03% ($CO_2$). Gas connections during calibration was 17.1% ($O_2$) and 5% ($CO_2$). The turbine flowmeter for the gas analyser was calibrated using a 3L calibration syringe (Hans Rudolph, inc., Kansas city, USA). Cardiac response, in the form of heart rate (HR), was measured using a portable HR monitor (Polar Electro, Kempele, Finland).

#### 2.2.5.2 Measuring cardiorespiratory response

Volume of $O_2$ ($VO_2$), volume of $CO_2$ ($VCO_2$), and HR were measured in rest one and rest nine for each trial. This allowed a comparison of cardiorespiratory response during recovery between the start and end of each trial between sexes. Given that participants SR their rest periods, $VO_2$, $VCO_2$ and HR rest one and rest nine was normalised as a percentage of total recovery time. With overall normalised average (100%) data or smoothing of the data and differentiating raw displacement data (cubic spline method (Vint & Hinrichs., 1996)), for the three measures, been used to compare between sexes, trials, and the start and end of each trial. Data was normalised as participants or sexes may have had a lower $VO_2$, $VCO_2$ and or HR due to a longer SR rest duration, which would allow a greater period of time for these measures to return closer to normal resting values (Yamagishi & Babraj., 2016). There is also evidence that indicates that participants would over-estimate their rest by at least 10% (Phillips, Thompson, Oliver., 2014). Therefore, removing the error of certain participants or sex experiencing a lower $VO_2$, $VOC_2$ and or HR value due to a longer SR rest duration. $VO_2$, $VCO_2$ and HR values were recorded at two sec averages, which may be less reliable than using longer sec averages for identifying the most processed data point (Robergs, Dwyer, Astorino., 2010). However, due to the uncertainty of when a participant would decide when to perform their next sprint this seemed appropriate.

### 2.2.6 Statistical analyses

Statistical analysis was analysed on IBM SPSS Version 22.0 software. To explore significance between sexes and trials a two-way (sex * trial) analysis of

variance (ANOVA) was used. The ANOVA compared the following: mean recovery time, MPO, and FI; the CS, participants' reliable trial (based on within-trial CV for MPO) based on the first four trials, 10% reduced rest (RR), and the 15% RR trial; physiological between R1 and R9 for the SR, 10% RR, and 15% RR trials. Statistical significance was set up as $p \leq 0.05$, and data are mean with ± standard deviation (SD). Significant main effects between trials were further explored by using Bonferroni post hoc analysis. Significant main effects between sexes and groups were further explored by using an independent samples T test. In the case of a significant interaction, the data was split by sex, the ANOVA test was performed again and significant main effects were explored as previously described. If sphericity was violated then Greenhouse-Geisser corrections were used. A Pearson's correlation was used to identify a link between trial data and cardiorespiratory data.

## 2.3 Results

### 2.3.1 Self-regulated rest:

Table 2.1 shows a comparison between all trials and best trial for SR recovery duration, MPO, FI and CV. A significant main effect was present between trials for SR recovery duration ($F_{6,113.262} = 2.967$, $p < 0.05$), post hoc indicates that trial 6 recovery time is significantly shorter than trial 4 ($p < 0.05$). No interaction was present in SR recovery ($F_{1, 20.044}, = 0.396$, $p > 0.05$). No significant main effect was present between sex in SR recovery time ($F_{1, 20.007} = 0.386$, $p > 0.05$).

### 2.3.2 MPO and MPO % change from the CS

A significant main effect was present between trials in MPO ($F_{7, 133.059}, = 4.117$, $p < 0.05$), post hoc indicates that trial 6 MPO data is significantly lower than the criterion sprint and trial 3 ($p < 0.05$). A significant main effect was also present between sex for trial MPO ($F_{1, 20.019}, = 24.612$, $p < 0.05$), post hoc also indicates that male MPO data is significantly higher compared to female MPO

across all trials ($p < 0.05$). An interaction was present in MPO ($F_{7, 133.051} = 2.065$, $p < 0.05$), female CS MPO is significantly higher than trials 1, 2, 4, best trial and 6 ($p < 0.05$). A significant main effect was present between trials in MPO % change from the CS ($F_{6, 112.97} = 4.279$, $p < 0.05$), post hoc indicates that trial 6 data is significantly greater than trials 3-5 and best SR trial ($p < 0.05$). A significant main effect of sex was also present in MPO % change from the CS ($F_{1, 19.839} = 8.021$, $p < 0.05$), post hoc indicates that female data is significantly greater than male data in trials 2-6 and best SR trial ($p < 0.05$). No significant interaction was present in MPO % change from the CS ($F_{6, 112.97} = 0.738$, $p > 0.05$).

### 2.3.3 FI and CV

FI was significantly different across trials ($F_{6, 112.049} = 2.237$, $p < 0.05$); post hoc indicates that trial 1 FI is significantly greater than best trial ($p < 0.05$). No significant main effect between sex ($F_{1, 18.405} = 0.015$, $p > 0.05$) or interaction ($F_{6, 112.086} = 1.516$, $p > 0.05$) was present for FI. A significant main effect between trials was present for CV ($F_{6, 111.696} = 3.886$, $p < 0.05$), post hoc indicates that the best trial was significantly lower than trials 1 and 2 ($p < 0.05$). No significant main effect of sex ($F_{1, 18.356} = 0.486$, $p > 0.05$) or significant interaction ($F_{6, 111.752} = 0.737$, $p > 0.05$) was present in CV.

Table 2.1: Performance variables across the 4 trials of self-regulated repeated sprint exercise for both sexes.

| | Criterion Sprint | Trial 1 | Trial 2 | Trial 3 | Trial 4 | Best Trial | Trial 5 | Trial 6 |
|---|---|---|---|---|---|---|---|---|
| Recovery time (sec) | | | | | | | | |
| Male | - | 88 ± 30 | 92 ± 31 | 98 ± 41 | 112 ± 48 | 101 ± 48 | 82 ± 29 | 77 ± 28‡ |
| Female | - | 102 ± 44 | 103 ± 40 | 102 ± 32 | 108 ± 44 | 105 ± 42 | 97 ± 29 | 88 ± 27‡ |
| Mean power output (W.kg $^{-1}$) | | | | | | | | |
| Male | 11.3 ± 1.3* | 11 ± 1.6* | 11.2 ± 1.3* | 11.5 ± 1.4* | 11.4 ± 1.2* | 11.4 ± 1.6* | 11.4 ± 1.4* | 11 ± 1.4*† |
| Female | 9.3 ± 1.2+ | 8.7 ± 1.3 | 8.5 ± 1.0 | 8.8 ± 1.1 | 8.7 ± 1 | 8.7 ± 1.1 | 8.9 ± 1.1 | 8.6 ± 1.4*† |
| Mean power output change to CS (%) | | | | | | | | |
| Male | - | -2.3 ± 9 | -0.1 ± 7.4 | 2.5 ± 7.9 | 1.1 ± 7.3 | 1.3 ± 6.6 | 2 ± 7 | -1.2 ± 8 |
| Female | - | -6.3 ± 8.3 | -8.1 ± 6.9 | -4.8 ± 5.9 | -5.7 ± 7.4 | -6.3 ± 6 | -4.5 ± 5.7 | -9.8 ± 8.6 |
| Fatigue index (%) | | | | | | | | |
| Male | - | 6.2 ± 4.6ϕ | 5.9 ± 3.7 | 6.9 ± 3.9 | 6.6 ± 4.8 | 4.7 ± 3.7 | 4.9 ± 3.9 | 5 ± 1.5 |
| Female | - | 8.7 ± 4.4ϕ | 8.7 ± 9 | 5.0 ± 1.9 | 5.3 ± 2.3 | 3.6 ± 1.3 | 4.4 ± 2.3 | 4.8 ± 2.2 |
| CV (%) | | | | | | | | |
| Male | - | 4.7 ± 3.8 | 3.5 ± 1.3 | 3.6 ± 1.8 | 3.9 ± 2.4 | 2.3 ± 0.7** | 2.4 ± 1.1 | 3.3 ± 1.5 |
| Female | - | 4.9 ± 2.4 | 5.1 ± 4.3 | 3.7 ± 1.0 | 3.7 ± 1.7 | 2.8 ± 1.1** | 3.1 ± 1.6 | 3.4 ± 1.5 |

*Table 2.1: ‡Significantly lower than trial 4. * Significantly greater than corresponding female data. † Significantly lower than trial 3 and criterion sprint.** Significantly lower than trials 1 and 2. + Interaction, female MPO CS data is significantly greater than trials 1, 2, 4 best trial and 6. ϕ significantly greater than best trial.*

### 2.3.4 $VO_2$, $VCO_2$ and HR resting data

Figure 2.1 (A) shows rest 1 and rest 9 normalised $VO_2$ data for the best SR trial, 10% RR and 15% RR trials for both sexes, (B) normalised $VO_2$ curve data in best SR trial for both sexes, (C) normalised $VO_2$ curve data in 10% RR trial for both sexes, and (D) normalised $VO_2$ curve data in 15% RR trial for both sexes. There was a significant main effect of mean normalised $VO_2$ data in trial/ rest period ($F_{5, 78}$, = 14.904, $p < 0.05$), post hoc indicates that all rest 9 data are significantly greater than all rest 1 data ($p < 0.05$). A significant main effect between sex was also present ($F_{1, 15.983,}$ = 70.228, $p < 0.05$), post hoc indicates that male $VO_2$ data is greater than female data across all trials and recovery periods ($p < 0.05$). A significant interaction (sex*trial) was also present ($F_{5, 78.093}$ = 2.647, $p < 0.05$), post hoc indicates that male SR rest 9 $VO_2$ data is significantly greater than all trial rest 1 data ($p < 0.05$).

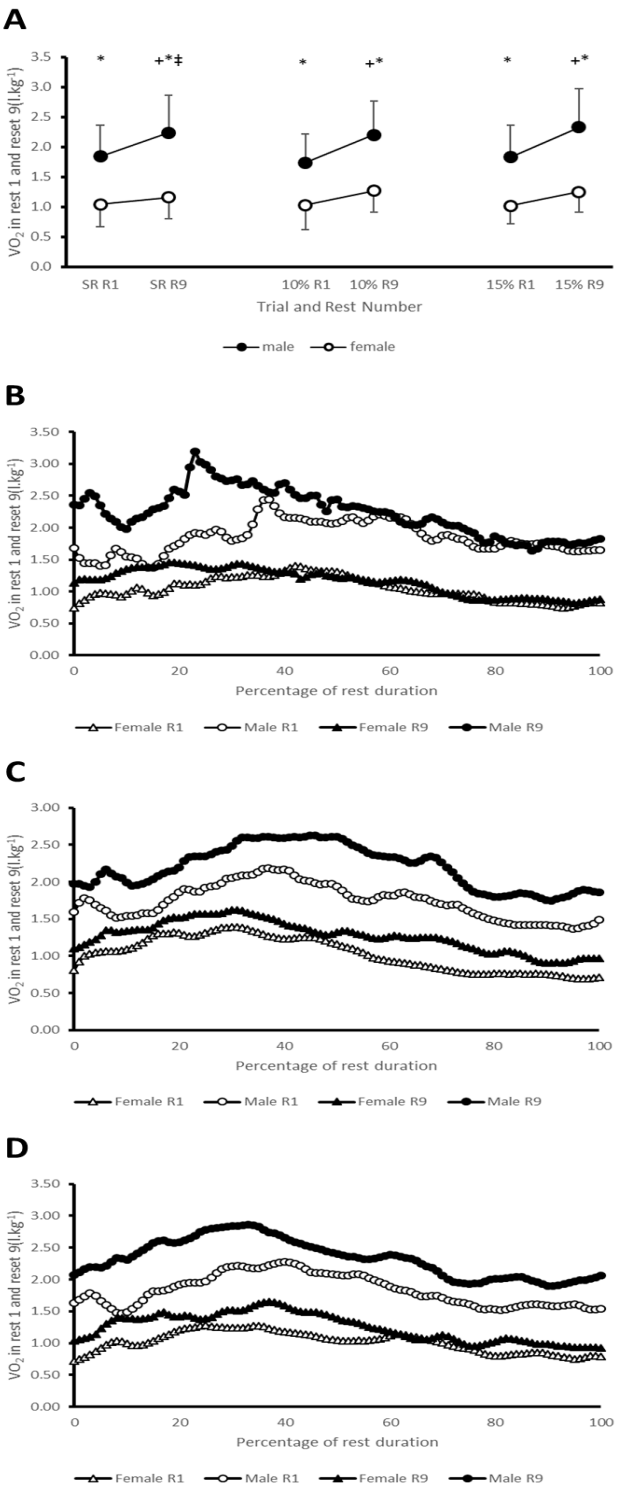


*Figure 2.1: (A) Normalised $VO_2$ in best SR trial, 10% and 15% RR trials, data shows rest 1 vs. rest 9, (B) best SR trial curve both sexes, (C) 10% RR trial curve both sexes, and (D) 15% RR trial curve both sexes. * Significantly greater than females ($p < 0.05$). + Significantly greater than all rest 1 data. ‡ Significant interaction, male SR R9 is greater than R1 data in all trials.*

Percentage of $VO_2$ peak during best SR trial, 10 and 15% RR trials is shown in table 2.2. No significant main effect of sex ($F_{1, 163.106,} = 2.279$, $p > 0.05$) or interaction was present ($F_{5, 78.205,} = 1.064$, $p > 0.05$). However, a significant main effect of rest number was present ($F_{5, 78.205,} = 16.05$, $p < 0.05$) with post hoc indicating that all rest 9 data was significantly greater than all rest 1 data ($p < 0.05$).

| Sex | SR R1 | SR R9 | 10% R1 | 10% R9 | 15% R1 | 15% R9 |
|---|---|---|---|---|---|---|
| Male | 54 ± 7 | 66 ± 16* | 51 ± 7 | 65 ± 11* | 54 ± 8 | 68 ± 13* |
| Female | 50 ± 11 | 55 ± 9* | 49 ± 13 | 61 ± 8* | 49 ± 7 | 59 ± 8* |

*Table 2.2: Percentage of $VO_2$ peak during rests 1 and 9 in best SR trial, 10 and 15% RR trials. * Significantly greater than all rest 1 data.*

Table 2.3 shows correlation values (r) comparing between the sum of MPO in each trial and the sum of $VO_2$ in each trial, and overall trials sum of MPO and the overall trials sum of $VO_2$. Significant correlations occur in SR MPO vs. SR $VO_2$ ($p < 0.05$), 10% RR MPO vs. 10% RR $VO_2$ ($p < 0.05$), 15% RR MPO vs. 15% RR $VO_2$ ($p < 0.05$), and overall MPO vs. overall $VO_2$ ($p < 0.05$).

| **Measure** | SR MPO vs. SR $VO_2$ | 10% RR MPO vs. 10% RR $VO_2$ | 15% RR MPO vs. 15% RR $VO_2$ | Overall MPO vs. Overall $VO_2$ |
|---|---|---|---|---|
| **Correlation** | r = 0.82* | r = 0.66* | r = 0.62* | r = 0.78* |

*Table 2.3: Correlation values comparing sum of MPO in each trial and the sum of $VO_2$ in each trial, and overall trials sum of MPO and the overall trials sum of $VO_2$. Overall MPO is a combination sum of MPO from SR, 10% and 15% RR trials. Overall $VO_2$ is a combination sum of normalised average $VO_2$ from rests 1 and 9 in SR, 10% and 15% RR trials. * Significant correlation ($p < 0.05$).*

Figure 2.2 (A) shows rest 1 and rest 9 normalised $VCO_2$ data for the best SR trial, 10% RR and 15% RR trials for both sexes, (B) normalised $VCO_2$ curve data in best SR trial for both sexes, (C) normalised $VCO_2$ curve data in 10% RR trial for both sexes, and (D) normalised $VCO_2$ curve data in 15% RR trial for

both sexes. There was a significant main effect of mean normalised $VCO_2$ data in trial/ rest period ($F_{5, 78.198}$, = 11.399, $p < 0.05$) and between sex ($F_{1, 16.057}$, = 53.715, $p < 0.05$). Post hoc indicates that rest 9 data in 10% and 15% RR trials is significantly greater than all rest 1 data, and SR rest 9 is significantly greater than SR rest 1 ($p < 0.05$). Post hoc between sex indicates that mean normalised $VCO_2$ is significantly greater in all trials and rest periods ($p < 0.05$). A significant interaction (sex*trial) was also present ($F_{5, 78.198} = 2.476$, $p < 0.05$), post hoc indicates that male 10% RR rest 9 is significantly greater than SR rest 1 and 10% RR rest 1 data, and male 15% RR rest 9 data is significantly greater than 10% RR rest 1 data ($p < 0.05$).

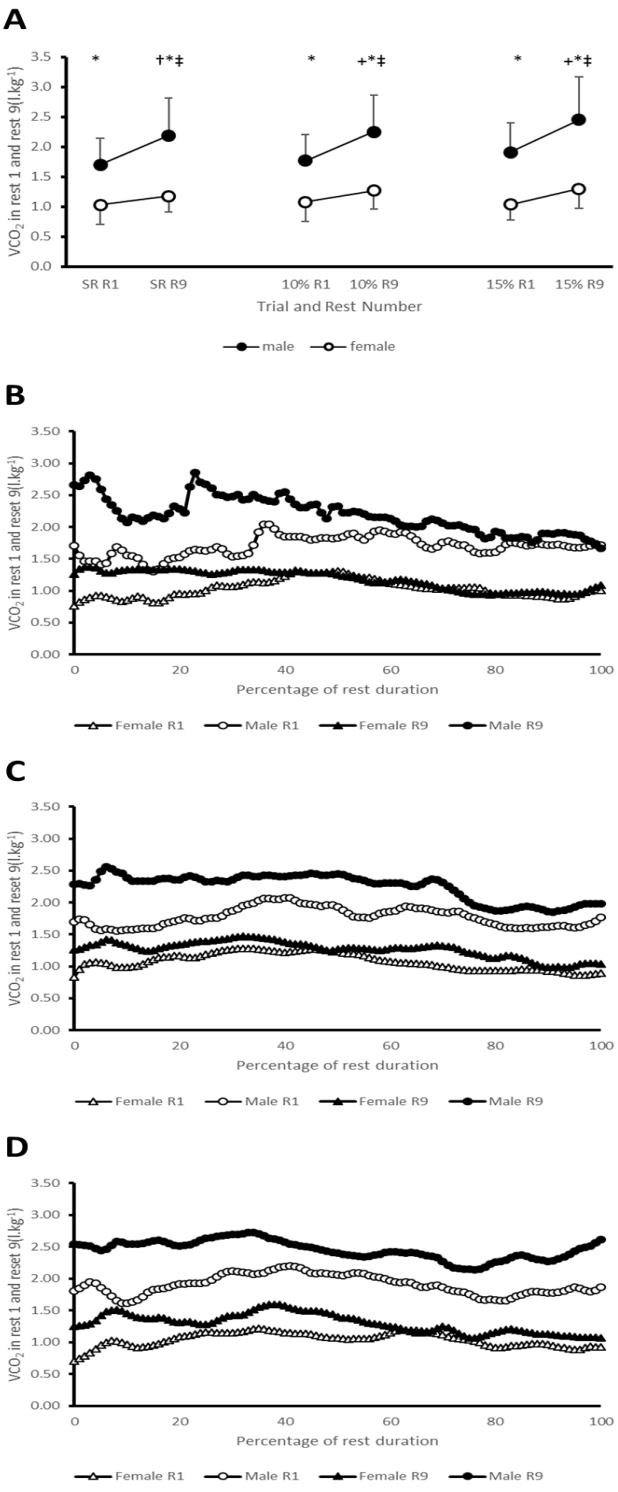


*Figure 2.2: (A) Normalised $VCO_2$ in best SR trial, 10% and 15% RR trials, data shows rest 1 vs. rest 9, (B) best SR trial curve both sexes, (C) 10% RR trial curve both sexes, and (D) 15% RR trial curve both sexes. * Significantly greater than females ($p < 0.05$). † Significantly greater than SR R1 data. + Significantly greater than R1 in all trials. ‡ Significant interaction, male 10% R9 is greater than 10% R1 and SR R1, and male 15% R9 is greater than 10% R1.*

Table 2.4 shows correlation values (r) comparing between the sum of MPO in each trial and the sum of $VCO_2$ in each trial, and overall trials sum of MPO and the overall trials sum of $VCO_2$. Significant correlations occur in SR MPO vs. SR $VCO_2$ ($p < 0.05$), 10% RR MPO vs. 10% RR $VCO_2$ ($p < 0.05$), 15% RR MPO vs. 15% RR $VCO_2$ ($p < 0.05$), and overall MPO vs. overall $VCO_2$ ($p < 0.05$).

| **Measure** | SR MPO vs. SR $VCO_2$ | 10% RR MPO vs. 10% RR $VCO_2$ | 15% RR MPO vs. 15% RR $VCO_2$ | Overall MPO vs. Overall $VCO_2$ |
|---|---|---|---|---|
| **Correlation** | r = 0.77* | r = 0.64* | r = 0.58* | r = 0.73* |

*Table 2.4: Correlation values comparing sum of MPO in each trial and the sum of $VCO_2$ in each trial, and overall trials sum of MPO and the overall trials sum of $VCO_2$. Overall MPO is a combination sum of MPO from SR, 10% and 15% RR trials. Overall $VCO_2$ is a combination sum of normalised average $VCO_2$ from rests 1 and 9 in SR, 10% and 15% RR trials. * Significant correlation ($p < 0.05$).*

Figure 2.3 (A) shows rest 1 and rest 9 normalised HR data for the best SR trial, 10% RR and 15% RR trials for both sexes, (B) normalised HR curve data in best SR trial for both sexes, (C) normalised HR curve data in 10% RR trial for both sexes, and (D) normalised HR curve data in 15% RR trial for both sexes. There was a significant main effect of mean normalised HR data in trial/ rest period ($F_{5, 75.424}$, = 31.566, $p < 0.05$), post hoc indicates that rest 9 data in all trials is significantly greater than all rest 1 data in all trials ($p < 0.05$). No significant main effect was present between sex ($F_{1, 16.158}$, = 0.473, $p > 0.05$) and so significant interaction (sex*trial) was present ($F_{5, 75.424}$, = 0.345, $p > 0.05$).

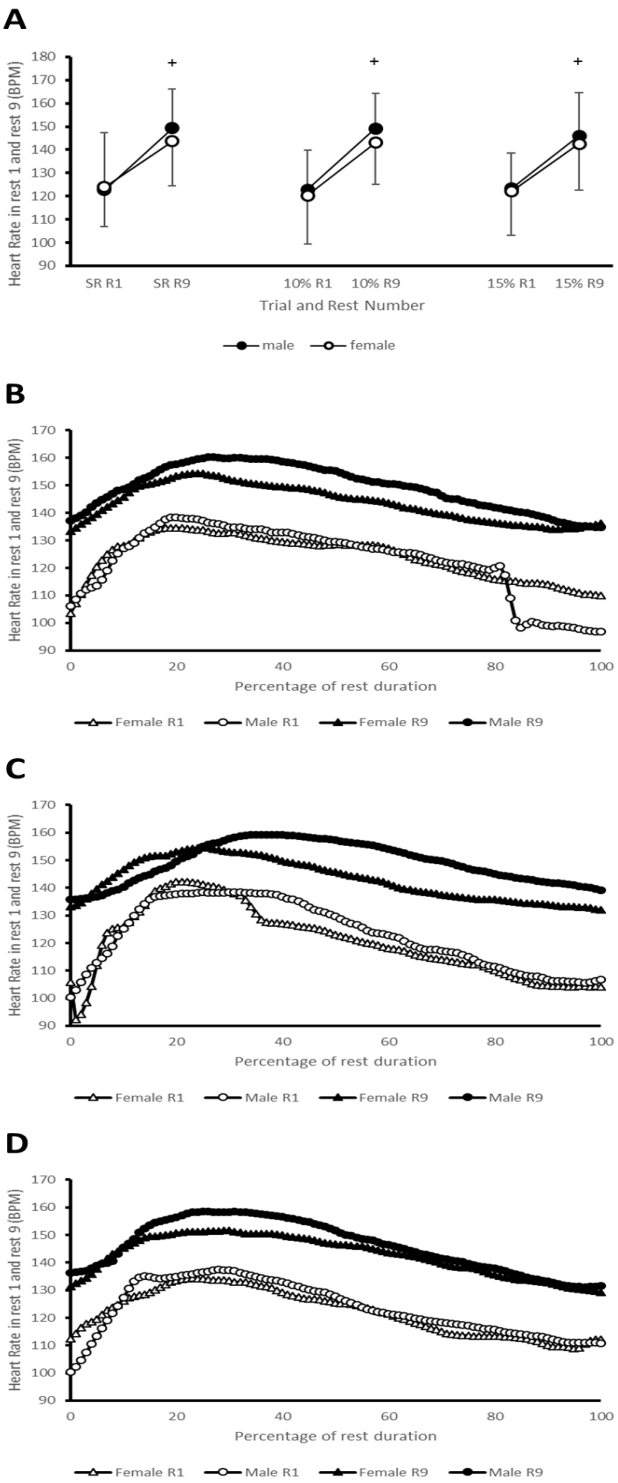


*Figure 2.3: (A) Normalised HR in best SR trial, 10% and 15% RR trials, data shows rest 1 vs. rest 9, (B) best SR trial curve both sexes, (C) 10% RR trial curve both sexes, and (D) 15% RR trial curve both sexes. + Significantly greater than all R1 data ($p < 0.05$).*

Table 2.5 shows correlation values (r) comparing between the sum of MPO in each trial and the sum of HR in each trial, and overall trials sum of MPO and the overall trials sum of HR. No significant correlation occurred ($p > 0.05$).

| **Measure** | SR MPO vs. SR HR | 10% RR MPO vs. 10% RR HR | 15% RR MPO vs. 15% RR HR | Overall MPO vs. Overall HR |
|---|---|---|---|---|
| **Correlation** | r = 0.22 | r = -0.06 | r = 0.21 | r = 0.3 |

*Table 2. 5: Correlation values comparing sum of MPO in each trial and the sum of HR in each trial, and overall trials sum of MPO and the overall trials sum of HR. Overall MPO is a combination sum of MPO from SR, 10% and 15% RR trials. Overall HR is a combination sum of normalised average HR from rests 1 and 9 in SR, 10% and 15% RR trials.*

## 2.4 Discussion

The aim of this study was to investigate the reliability and accuracy of SR repeated sprint performance in males and females. It was found that males require a larger consumption of $VO_2$ and have a higher $VCO_2$ output compared to females during SR recovery. FI and CV was not influenced by reducing recovery time. However, MPO performance was impaired for both sexes, MPO data from trial 6 (15% RR) is significantly less than the CS (males = -1.2 ± 8%, females = -9.8 ± 8.6%) and trial 3 (males = 2.5 ± 7.9%, females -4.8 ± 5.9%). A significant interaction in MPO (female MPO CS data is significantly higher than trials 1-2, 4, 6 and best SR trial) suggests that the main effect MPO data is caused by a pacing strategy being employed by females that results in a drop in MPO during sessions to maintain consistency. For example, when female participants know the number of sprints to be performed, muscle activation levels are lower than when they think they are doing half that number (Billuat et al., 2011).

No significant difference was found between sexes for SR rest duration (Table 2.1). It has been suggested that females would require a shorter SR rest

duration due to the predominance of type I muscle fibres, a larger use of fat oxidation and possibly greater PCr recovery when compared to males (Esbjornsson-Lijedahl, Bodin, Jansson., 2002; Kent-Braun & Alexander., 2000; Laurent et al., 2010; Roepstorff et al., 2006**)**. The metabolic and morphological profile of females can reduce their fatigability during high intensity cycle sprints compared to males, which would be expected to translate into shorter recovery duration (Esbjornsson-Lijedahl, Bodin, Jansson., 2002; Kent-Braun & Alexander., 2000; Laurent et al., 2010; Perez Gomez et al., 2008; Roepstorff et al., 2006). However, we see similar SR rest times which may be due to the high intensity and cycling exercise selection (Hunter., 2014; Knetchtle et al., 2004; Laurent et al., 2010). Knetchtle et al., (2004) found that both sexes experience similar lactate levels when cycling at 55% (male: ~1.11, female: ~1.2 mmol x $l^{-1}$), 65% (male: ~1.48, female: ~1.49 mmol x $l^{-1}$) and 75% (male: ~2.15, female: ~2.11 mmol x $l^{-1}$) of their $VO_2$ max. However, males have a significantly higher amount of carbohydrate (CHO) oxidation than females when cycling at any of the $VO_2$ max percentages (55%: male > 100, female > 80; 65%: male > 130, female > 90; 75%: male > 145, female > 105 cal*$min^{-1}$*$kg^{-1}$ (Knetchtle et al., 2004)). There is also no significant sex difference in fat oxidation at 75% $VO_2$ max in cycling (Knetchtle et al., 2004). Therefore, repeat sprint activity (RSA) may remove a female dominance in resisting peripheral fatigue at lower exercise intensities (Knetchtle et al., 2004), resulting in similar rest periods being taken between sexes.

Males produced a significant larger amount of MPO across all trials (Table 2.1). It has previously been found that when given an insufficient rest period (30sec) males experience a larger decrease in power during repeat sprint training compared to females (Billaut & Bishop., 2012). Potentially due to greater anaerobic glycolysis leading to inorganic phosphate accumulation (Gaitanos et al., 1993; Li et al., 2002). When taking into account both sexes, no significant difference was found for MPO data between SR trials and 10% RR trial. This is consistent with previous research that male participants over-estimate their required SR recovery by at least 10% (Phillips, Thompson, Oliver., 2014). However, the current study found that not all participants (2 males, 1 female) could successfully maintain MPO between trials 1-4 with a further female

participant been unsuccessful at trial 5. Indicating that using SR rest to maintain MPO is not as reliable as previously documented (Phillips, Thompson, Oliver., 2014). The current study also found a significant decline in MPO when SR rest was reduced by 15% (trial 6) for both sexes (Table 2.1). Post hoc data indicates that the CS and trial 3 MPO is significantly greater than trial 6 for both sexes. Therefore, data suggests that the majority (85%) of physically active participants over-estimate their SR recovery duration by at least 10% but not more than 15%. Female MPO was decreased significantly compared to male MPO when SR rest was reduced by 15% and compared to the CS (males: -1.2 ± 8%, females: -9.8 ± 8.6%, $p < 0.05$). The rate of FI is similar across sexes (Table 2.1) which suggests that the rate of decline in power production is not different. FI is only significantly greater in trial 1 when compared to the participants' best trial. Similar results occurred with CV with no difference between sex and trials 1-6, but the participants' best trial CV was significantly less compared to trials 1-2 CV. However, MPO data has a significant interaction (sex*trial) that indicates that female MPO in trials 1-2, 4, 6 and best trial is significantly lower than the female CS. Given that female FI and CV is unaffected, it could suggest that females are adopting pacing strategies during trials 1-6. Pacing has previously been found within cycling sprint activity using a variation in sprint durations (5, 15, 30 and 45 sec (Wittekind, Micklewright, Beneke., 2011)). Wittekind, Micklewright, Beneke., (2011) found that peak power was significantly greater using a 5 sec sprint compared to 15, 30 and 45 sec sprints, and peak power was also significantly greater using a 15 sec sprint compared to 30 and 45 sec sprints, despite peak power been achieved between 0-10 sec of a sprint (Vandewalle, Pérès, Monod., 1987). What may have happened within the present study is that females may have paced their efforts due to having to maintain their MPO from the CS for ten sprints, which may be a similar effect from the findings of Wittekind, Micklewright, Beneke., (2011). Females may employ a greater amount of pacing strategies or adjusted their approach after the CS due to cycling against 7.5% of their BM. This may be because 7.5% for females is a greater relative intensity due to their lower muscle mass and greater fat mass (Perez Gomez et al., 2008). Suggesting why CV and FI were not affected in trials 1-6 for females despite percentage change of MPO compared to the CS was always lower (females: -4.8 – -9.8%

difference, males: -2.3 – 2% difference (Table 2.1)). Therefore, females may require a reduction in resistance to cycle against in order to maintain their CS MPO over ten sprints. It would appear that using CV may not be a reliable indicator for identifying if a participant is successful at maintaining power output or speed as previously documented (Glasiter et al., 2010; Phillips, Thompson, Oliver., 2014). Therefore, comparing percentage change between the CS and MPO may be a better indicator for successfully maintaining MPO during RSA compared to CV as previously used (Glasiter et al., 2010; Phillips, Thompson, Oliver., 2014).

$VO_2$ and $VCO_2$ R9 data is significantly greater than all R1 data (best SR, 10% and 15% RR trials) for both sexes, suggesting greater aerobic demand for recovery from later sprints in both sexes (Figure 2.1 and 2.2). Normalised gas data suggests that male participants experience a greater aerobic demand compared to females during SR rest and reduced SR rest (10 and 15%). However, Table 2.2 indicates that this greater aerobic demand is due to the higher $VO_2$ peak in males (43 $ml.kg^{-1}.min^{-1}$) than in females (33 $ml.kg^{-1}.min^{-1}$). With Table 2.2 showing no significant difference in percentage of $VO_2$ peak during rests 1 and 9 for all the measured trials between sexes. Indicating that there may be no sex difference in regards to aerobic response during SR repeat sprint cycle activity. The significant increase from rest 1 to rest 9 in all measured trials for $VO_2$, $VCO_2$ and HR in both sexes could suggest that PCr is recovered at a similar rate due to an increase in oxidative phosphorylation (McCartney et al., 1986; Spriet et al., 1989). Which is in keeping with a greater ATP turnover to regenerate maximal effort (McCartney et al., 1986; Spriet et al., 1989), and could suggest why SR rest duration is similar between sexes. Correlation data (Table 2.3, 2.4) indicates that a greater sum of MPO in the trials is significantly linked with an increase in $VO_2$ and $VCO_2$ during the recovery for both sexes. This significant correlation further indicates SR repeat sprint cycling leads to an increased use of aerobic metabolism in order to recover PCr and repeatedly generate maximal MPO (McCartney et al., 1986; Spriet et al., 1989). The correlation between the gas measures and sum of MPO is at its strongest during the best SR trial ($VO_2$: $r = 0.82$, $VCO_2$: $r = 0.77$), and at its weakest during the 15% RR trial ($VO_2$: $r = 0.62$, $VCO_2$: $r = 0.58$). The drop in correlation

strength in the 15% RR trial could be a reflection of both sexes dropping their MPO (males: ~-1.2%, females: ~-9.8%). Despite a drop in MPO in the 15% RR trial percentage of $VO_2$ peak still increases by ~14% and ~10% in males and females respectively between rests 1 and 9. This could be due to a shorter work:rest ratio in the 15% RR trial which is thought to create a greater aerobic response during repeat sprint cycling (Kavaliauskas, Aspe, Babraj., 2015). Furthermore, the correlation data could indicate that a greater $VO_2$ peak may be vital in maintaining MPO whilst using SR rest. Therefore, females or participants with a lower $VO_2$ peak may require a reduction in resistance (< 7.5% BM) to maintain their MPO through SR rest. This may remove the possible pacing tactics that may have occurred within the female participants in this study.

## 2.5 Limitations

The current study indicates that females are unable to maintain their CS MPO across ten sprints as well as males, with males producing on average a MPO ~2.5% greater than their CS in trial 3. The current method for measuring CS appears to be only used by Phillips, Thompson, Oliver., (2014) and there is limited research to indicate if this CS procedure has been used previously. It is thought that a learning effect occurs for participants when they perform repeat cycle sprints due to a decrease in CV measures for power output after two trials (CV = 0.5-1.9%) compared to completing a third trial (CV = 0.3-0.7% (Hopkins, Schabort, Hawley., 2001)). The higher CV values typically found within the first two trials of repeated cycle sprints could indicate that the 5% limit used in the CS procedure could have error measurement of 0.5-1.9% below or above the 5% cut off. Not knowing how much accurate measurement of error may be present in the 5% CS cut off may have affected the percentage change between the CS and MPO trial average for both males and females (Table 2.1). Therefore, it is possible that males maintained a MPO closer to their CS in each trial compared to females due to an inaccurate measure of the CS. Identifying an accurate measurement of error in the CS would lead to more reliable data for percentage change between the CS and trial average MPO.

During the current study, a number of participants complained about the use of the gas mask, with some participants claiming it made them feel claustrophobic. Another regular complaint from the participants was the level of discomfort they experienced from the bike seat. It is possible that these two factors affected the SR rest duration, by possibly selecting a shorter rest period in order to remove themselves quicker from the claustrophobic and discomfort conditions.

## 2.6 Conclusion

In conclusion, this study shows less reliability of participants maintaining power output than previously documented (Phillips, Thompson, Oliver., 2014). 95% of participants that could SR their rest to maintain MPO over-estimated their SR rest by 10%, which is similar to the finds of Phillips, Thompson, Oliver., (2014). However, reducing rest by 15% leads to a loss in power production, more so in females than in males. Using 7.5% body mass as a resistance might play a key factor in why females cannot maintain power output as greatly as males. There is also the possibility that females might be pacing their efforts to protect themselves from homeostatic harm. Further research should identify if reducing the resistance allows females to better SR their rest to maintain MPO. Using CV appears to be an unreliable tool for identifying maintenance of MPO. Instead, comparing the percentage change of each MPO in a trial against the CS could be more reliable. Further research should also examine if SR RSA leads to any performance adaptations and verify if it can be a useful tool for team sport athletes as previously advised (Glaister et al., 2010; Phillips, Thompson, Oliver., 2014).

## 2.7 Practical implications

This present study is not consistent with previous research (Glaister et al., 2010; Phillips Thompson, Oliver., 2014) and that not all participants can SR their rest as a reliable tool for maintaining maximal repeat sprint performance, with females appearing to pace their efforts more than males when attempting to maintain CS MPO. This study has identified 4 practical implications. 1) SR rest duration varies between physically active participants and is personal to

each individual; athletes should not be influenced by a teammate's ability to SR faster or longer. 2) There is a strong likelihood that SR recovery during power testing would not see a decrease in performance when compared to using fixed rest periods, and would allow a coach to gain accurate MPO data from each athlete. 3) Athletes performing repeat sprint cycle training using SR rest between sprints could see an improvement in their neurological response, leading to a faster sprint speed due to an improved power output, by repeatedly producing maximal effort. Due to the use of longer work to rest ratios during high intensity training (Kavaliauskas, Aspe, Babraj., 2015). 4) SR rest periods do not replicate the short rest periods typically found within team sports. However, males significantly increase their aerobic demand during SR RSA which could be a factor for improving endurance (Kavaliauskas, Aspe, Babraj., 2015). The 10% overestimated SR rest may affect the required aerobic demand during RSA that leads to improvements in endurance. Therefore, coaches should be aware that improvements in endurance might not occur when using SR rest during repeat sprint training. Coaches should understand the potential benefits and limitations SR rest can offer athletes, and that the data created from this study should be treated as population specific until further research has been conducted with an elite athlete population. This will identify if elite athletes express similar responses to SR RSA.

## 2.8 Proceeding research

The data from the present study suggests that males can SR their rest in order to maintain their MPO greater than females. Males also experience a drop in their MPO (-1.2 ± 8% compared to CS) when SR rest is reduced by 15%, however females experience a greater drop in their MPO (-9.8 ± 8.6% compared to CS) when SR rest is reduced by 15%. Therefore, the proceeding research will use male participants to identify the effects of using SR rest during HIT, given that the maintenance of power output in HIT is a potential key factor to increasing endurance and power output adaptations (Hazell et al., 2010; Lloyd Jones, Morris, Jakeman., 2017). It is unsure if using SR rest during HIT will lead to any positive performance adaptation due to the over-estimation in SR rest (Phillips, Thompson, Oliver., 2014). However, despite the over-

estimation in SR rest, repeated sprints with a greater work:rest ratio will still experience an increase in aerobic demand in sprints as long as there are multiple sprint bouts (Hazell et al., 2010), which is thought to be a key factor for improving endurance measures (Kavaliauskas, Aspe, Babraj., 2015). However, Kavaliauskas, Aspe, Babraj., (2015) demonstrate that reducing the rest duration in HIT leads to greater endurance adaptations and increasing the rest duration leads to greater power output adaptations. Therefore, the proceeding study will identify if using SR rest during HIT will lead to any positive endurance and or power output adaptations, and compare against using a fixed rest (30 sec, 1:5 work to rest ratio) in HIT.

# 3 Chapter 3 – Study 2 (Training adaptations with self-recovery compared to fixed rest during sprint interval training)

## 3.1 Introduction

### 3.1.1 Maintenance of power

There are many studies that suggest that the maintenance of power during training is an important contributing factor for improving power output in athletes (Baechle & Earle., 2008; Creer et al., 2004; Hazel et al., 2010; Lloyd Jones, Morris and Jakeman., 2017; Kavaliauskas, Aspe, and Babraj., 2015). Maintaining power allows for a larger response from anaerobic pathways, with an increase in PCr and glycogen degradation, and greater neural adaptions such as improvement in motor unit recruitment, firing rate and synchronization post intervention (Creer et al., 2004; Kraemer, Fleck, Evans., 1996; Rodas et al., 2000). High intensity training (HIT), in the form of repeated cycle sprints (4-6 x 30sec sprint with 4min recovery, 8 sessions), has found to increase motor unit activation (Creer et al., 2004), with an increase in root mean squared (~27.7%) and decrease median frequency (~-16.8%) during electromyography (EMG) readings within the vastus lateralis muscle (Creer et al., 2004). Creer et al., (2004) also found significant increases in peak (~6%) and mean (~6%) power output post HIT. Within strength and power training, recruitment of type II muscle fibres are essential for maximal efforts and for increasing strength (Kraemer, Fleck, Evans., 1996). A number of studies using varied HIT protocols (4-6 x 30sec, 4-6 x 10 sec, 6 x 10sec sprints against 7.5% body mass) have reported increased power output post training (Creer et al., 2004; Hazell et al., 2010; Kavaliauskas, Aspe, and Babraj., 2015). It has been suggested that the maintenance of power during the training is a contributory factor in this adaptation (Hazell et al., 2010; Lloyd Jones, Morris, Jakeman., 2017; Yamagashi & Babraj., 2017).

Glaister et al., (2010) and Phillips Thompson, Oliver., (2014) suggest that physically active young adult male participants can reliably maintain multiple running sprint times and MPO during cycle sprints. This was achieved when participants self-regulated (SR) their own recovery periods when performing 12

x 30m sprints (Glaister et al., 2010) or 10 x 6 sec cycle sprints (7.5% body mass resistance (Phillips Thompson, Oliver., 2014 and Study 1)). However, Phillips Thompson, Oliver., (2014) and Study 1 found that physically active participants over-estimate their required recovery by at least 10%. In contrast to Glasiter et al., (2010) and Phillips Thompson, Oliver., (2014), Study 1 found that not all participants (2 males, 1 female) could maintain MPO, and females' average trial MPO was significantly less than their criterion sprint MPO. Indicating that the reliability of maintaining MPO using SR rest is not as reliable as previously indicated. Seiler & Hetlelid., (2005) found that SR rest (118 ± 23 sec), 1 min rest, 2 min rest and 4 min rest between running sprint bouts (6 x 4 min) had similar effects in terms of the blood lactate rise post sprint. Running velocity increased in sprints following 2 min and SR rest compared to 1 min rest. $VO_2$ during sprints was significantly higher for 2 min and SR rest compared to both 1 min and 4 min rest. However, these studies have only looked at the acute response during a single session and have not looked at training adaptions with SR. It has been suggested recently that work to rest ratio could affect the adaption to repeated sprint training, either in terms of power production or endurance adaptions (Kavaliauskas, Aspe, Babraj., 2015).

### 3.1.2 Improving power:

HIT has been shown to promote increases in both PPO (the highest watt value typically during the first 5-10 seconds of a sprint) and MPO (the overall mean average watt value during the sprint (Burgomaster et al., 2005; Creer et al., 2004; Hazell et al., 2010; Kavaliasukas, Aspe, Babraj., 2015; Rodas et al 2000)). HIT has been shown to increase PCr stores after training, thus allowing a faster resynthesize of ATP anaerobically in response to an explosive movement (Gaitanos et al., 1993; Rodas et al., 2000). Therefore, allowing larger sustainability of MPO and a larger PPO (Rodas et al., 2000; Laursen and Jenkins., 2002). The greater PCr stores are thought to be due to delaying the onset of a lower pH (~ 0.5 units) in skeletal muscle following training (Balsom et al., 1992; MacLaren & Morton., 2012). A build up in Pi and reduction in calcium ($Ca^{2+}$) sensitivity are thought to be causes for peripheral fatigue due to high rates of PCr hydrolysis (Westerblad, Allen, Lannergren., 2002). These potential

fatiguing factors prevent PCr resynthesis (Westerblad, Allen, Lannergren., 2002). Increasing PCr stores leads to the reduction in Pi, and the release and reabsorption of $Ca^{2+}$ within the sarcoplasmic reticulum is greater (Westerblad, Allen, Lannergren., 2002). HIT has been found to increase glycolytic enzyme activity (phosphofructokinase, lactate dehydrogenase) and increase muscle glycogen concentration (Rodas et al., 2000). The increased resting and during exercise stores of muscle glycogen is regulated by an increase in the glycolytic flux mechanism (MacLaren & Morton., 2012). The glycolytic flux is regulated by glucokinase activity which is a glucose sensor and increases blood glucose levels during exercise (MacLaren & Morton., 2012). Increasing phosphofructokinase and fructobisphosphatase activity increases the glycolytic shuttle and promotes insulin secretion from the pancreas (MacLaren & Morton., 2012), and contributes to ~40% ATP turnover during the first 15sec of a 30sec sprint (Parolin et all., 1999). Decreases in glycogen stores lead to an impairment of $Ca^{2+}$ release from the SR (Ørtenblad et al., 2011), therefore reducing the rate of muscular contraction and decreasing force production (Ørtenblad et al., 2001; Westerblad, Allen, Lannergren., 2002). Lactate dehydrogenase and acetyl coenzyme A (acetyl CoA) are responsible for improving the rate of conversion of lactate back to pyruvate (MacLaren & Morton., 2012). Allowing a greater transport of glycogenic intermediates to the Krebs cycle and the resynthesis of ATP aerobically (MacLaren & Morton., 2012). PPO can also increase due to the remodelling of myofibrils following high intensity resistance training (Seynnes, de Boer, Narici., 2007). Remodelling of the myofibrils can occur after one eccentric session which causes lengthening of the myofibrils (Yu, Carlsson, Thornell., 2004). It has also been demonstrated that an increase in myofibril (> ~ 2% $d^{-1}$) and sarcoplasmic (> ~ 2% $d^{-1}$) protein synthesis occurs after one session of HIT (10 x 1min sprints at ~ 95% HR max), and remains elevated (myofibril ~ 2% $d^{-1}$, sarcoplasmic > ~ 1.5% $d^{-1}$) 24 hours after (Bell et al., 2015). Proteins, sirtuin 1 and mitochondrial transcription factor A, located within the myofibrils are responsible for mitochondrial biogenesis (Little et al., 2010). It has also been found that HIT leads to an increase in sirtuin 1, glycogen depletion, and mitochondrial transcription factor A proteins, that in turn increases mitochondrial biogenesis (Camera, Hawley, Coffey., 2015; Little et al., 2010). With an increase in mitochondrial biogenesis this leads to an

increase in ATP production through aerobic metabolism and therefore produce a larger amount of energy during exercise (MacLaren & Morton., 2012). Therefore, given that HIT leads to improvements in PPO and MPO (Burgomaster et al., 2005; Creer et al., 2004; Hazell et al., 2010; Kavaliasukas, Aspe, Babraj., 2015; Rodas et al 2000), literature would suggest that the increases in sirtuin 1 and mitochondrial transpiration factor A proteins could be partly responsible for the increase in PPO and MPO after HIT.

It has also been demonstrated that HIT causes changes in muscle fibre distribution, change in $Ca^{2+}$ dynamics and alters neuromuscular function (Creer et al., 2004; Esbjörnsson et al., 1993; Ørtenblad et al., 2000; Pette., 1985; Pette., 1998; Pette & Staron., 1997; Ross & Leveritt., 2001). HIT (4-6 30sec sprints 4min recovery against 7.5% body mass,15 x 10sec sprints with 50sec recovery against 7% body mass, 2-6 x 15 and 30sec sprints against 7.5% body mass, 4-6 x 30sec sprints 15-20min recovery against 75g per kg body mass), has previously demonstrated a change in dominance in muscle fibre recruitment during exercise (Allemeier et al., 1994; Esbjörnsson et al., 1993; Jacobs et al., 1987; Jansson et al., 1990). Alterations have shown to decrease the recruitment of faster type muscle fibres (type IIX) and increase the recruitment of intermediate fast twitch muscle fibres (type IIA (Allemeier et al., 1994; Esbjörnsson et al., 1993; Jacobs et al., 1987; Jansson et al., 1990; Pette., 1998; Pette & Staron., 1997; Ross & Leveritt., 2001)). This also leads to a decrease in type I muscle fibre recruitment and in some cases shows no alteration in the recruitment of type I (Allemeier et al., 1994; Jacobs et al., 1987; Jansson et al., 1990; Pette., 1997; Ross & Leveritt., 2001). Neural impulse responses are responsible for these alterations, due to the progression/ loading of HIT, which alters metabolic homeostasis (Pette., 1985). Further neural adaptations, following 4 weeks HIT (4-10 x 30sec sprints with 4min recovery, twice a week), increased motor unit activation in the vastus lateralis (Creer et al., 2004). Which is in keeping with producing greater amounts of PPO, MPO and total work (Creer et al., 2004). It is also thought that following 5 weeks of HIT (20 x 10sec sprint with 50sec recovery, 3 times a week, against 8-8.5% body mass) leads to an increase in sarcoplasmic reticulum, due to an increase in $Ca^{2+}$ release of ~5.5% (Ørtenblad et al., 2000). Who also found greater maintenance of MPO

during 10 sprints in pre to post and compared to the control group (Ørtenblad et al., 2000).

It is unclear how important manipulating the work to rest ratio of multiple sprint bouts is for manipulating the training induced improvements in power (Hazell et al., 2010; Kavaliasukas, Aspe, Babraj., 2015; Yamagishi and Babraj., 2017). Kavaliasukas, Aspe, Babraj., (2015) found significant improvements in MPO, with a 1:8 (6 x 10sec sprint 80sec recovery, against 7.5% body mass), 1:12 (6 x 10sec sprint 120sec recovery, against 7.5% body mass) work to rest ratio, and PPO with the 1:8 work to rest ratio, following 6 sessions of HIT. Hazell et al., (2010) also show a significant increase in MPO, with a 1:8 (4-6 x 30sec sprint with 4min recovery against 7.5% body mass) and a 1:24 (4-6 x 10sec sprint with 4min recovery, against 7.5% body mass) work to rest ratio. They also found a significant increase in PPO with a 1:8, 1:24 and 1:12 (4-6 x 10sec sprint with 2min recovery against 7.5% body mass) work to rest ratio, following 6 sessions of HIT. Kavaliasukas, Aspe, Babraj., (2015) state that maintaining PPO in training, with higher work to rest ratios, leads to neuromuscular adaptations to create a larger training effect of power production. However, Hazell et al., (2010) found that the 1:8 work ratio group PPO, MPO and minimum power output during training was significantly lower when compared to the 1:24 and 1:12 work ratio groups. All three of their training groups found significant improvements in PPO regardless of work to rest ratio, with the 1:12 group been the only group not to improve MPO significantly. Causing debate whether maintaining power output during training is vital or not.

It has been found that sprint duration between 6-30 sec sees a shift from a dominance in anaerobic metabolism (glycolysis and PCr) to a dominance in aerobic metabolism as the duration of the sprint increases (Bogdanis et al., 1996; Bogdanis et al., 1998; Gaitanos et al., 1993; Parolin et al., 1999). Protocols using sprint bout durations of 6-30 sec have seen significant improvements in PPO and MPO (Hazell et al., 2010; Jakeman, Adamson, Babraj., 2012; Kavaliasukas, Aspe, Babraj., 2015; Yamagishi and Babraj., 2017). Fuel consumption during HIT has been previously documented, and potentially explains how participants improve in endurance testing (Gaitanos et

al., 1993; Bogdanis et al., 1996). Gaitanos used 10 x 6 sec sprints with a 30 sec recovery and found a decrease in MPO after each sprint. When comparing muscle biopsies, from sprint 1 and sprint 10, glycolysis and ATP utilisation had dropped from 44.1% - 16.1% and 6.3% - 3.8% respectively. Whereas, PCr increased from 49.6% - 80.1%, suggesting that the power output in the latter sprints were fuelled by an increase in oxidative metabolism (Gaitanos et al., 1993). However, the oxidative metabolism was not directly measured. Bogdanis et al., (1996) performed a similar study but identified changes in two 30 sec sprints separated by 4 min rest, measuring glycolysis, ATP + PCr, and aerobic contribution during the sprint. The findings of Bogdanis et al., (1996) supports the suggestion from Gaitanos et al., (1993) that a larger aerobic contribution occurs even after one sprint. They found that ATP turnover in glycolysis decreased from 48% - 36%, PCr + ATP also decreased from 23% - 20%, and aerobic contribution increased from 29% - 43%. This increase in aerobic contribution during HIT is thought to be a factor for improving aerobic capacity (Gaitanos et al., 1993; Gosselin et al., 2012). This shift to a dominance in aerobic metabolism is in keeping with a reduced force development (Russ et al., 2005).

### 3.1.3 Improving endurance

It is well documented that aerobic metabolism can be enhanced by repeated bouts of intense exercise (Hazell et al., 2010; Jakeman, Adamson, Babraj., 2010; Kavaliasukas, Aspe, Babraj., 2015; Rodas et al., 2000; Yamagishi and Babraj., 2017). More specifically HIT has found to increase the sarcolemmal lactate transport capacity, with an enhanced content of $MCT_1$ and $MCT_4$ (Pilegaard et al., 1999). Following 10 x 6 sec sprints there is a rightward shift in the blood lactate curve during an incremental time to exhaustion test (Jakeman Adamson, Babraj., 2012). Suggesting either altered lactate production or altered lactate utilisation. Increasing lactate transporter activity is known to improve recovery during moderate and high intensity exercise (Juel & Halestrap., 1999). Increased lactate transporter activity is suggested to be a key factor for improvements in time trial, time to exhaustion and critical power following HIT (Hazell et al., 2010; Jakeman, Adamson, Babraj., 2012; Kavaliauskas, Aspe,

Babraj., 2016; Yamagishi, Babraj., 2017). This enables a greater movement through the lactate shuttle, which maintains lower blood lactate concentration at the same absolute workload (Jacobs et al., 2011) and reflects increased lactate utilisation (Rodas et al., 2000). During HIT, skeletal muscle produces high volumes of lactate and pH levels drop by ~ 0.5 units (MacLaren & Morton., 2012); however, the type 1 fibres use lactate as a respiratory fuel (Juel & Halestrap., 1999). This is reflected in the findings in Juel et al., (2003) who found an increase in lactate and $H^+$ release in trained (8 weeks of maximal intensity leg extensor activity) vs. untrained participants, during a 30 W test. Along with increases in lactate and $H^+$ release, Juel et al., (2003) also found an increase in monocarboxylate transport protein 1 ($MTC_1$), $Na^+/H^+$ exchanger protein (NHE1) and an increased blood flow of ~16% within trained participants. Blood volume increases by an increase in lactate concentration and lactate uptake in skeletal muscle tissue (Gladden., 2000). As lactate increases 75-80% of the lactate is oxidised with the remaining 25-20% been converted into glucose and glycogen (Brooks., 2000). Therefore, allowing a greater ATP turnover to allow higher muscular contraction rates (Bogdanis et al., 1996; Gaitanos et al., 1993). Pilegaard et al., (1999), found similar results to Juel et al., (2003) but also found an increase in $MCT_4$ activity when comparing trained against untrailed participants. $MCT_4$ has similar purposes to $MCT_1$ but works directly with the removal of lactate within type II muscle fibres, so $MTC_4$ aids with oxidative and glycolic metabolism (Pilegaard et al., 1999). Therefore, fatiguing metabolites that become present with an increase in lactate (Pi and predominately within type II muscle fibres) are transported to the TCA cycle, via lactate transporter proteins $MCT_1$, $MCT_4$ and, at an increased rate in trained athlete's vs non-trained (Juel & Halestrap., 1999; Pilegaard et al., 1999). This would suggest that athletes would see an improvement in endurance tests that involve an increase in intensity with time, e.g. time to exhaustion test, due to an increase in $MTC_4$ leading to greater substrate provision to the TCA cycle. Following HIT there is an increase in enzymatic activity related to mitochondrial aerobic metabolism, citrate synthase and 3-hydroxyacyl-CoA dehydrogenase (HAD) (Rodas et al., 2000). These are oxidative mitochondrial proteins, with citrate synthase and HAD been responsible for the first and third step within the Kreb cycle respectively (MacLaren & Morton., 2012). Burgomaster et al., (2005)

and Shepley et al., (1992) found an increase in CS of ~ 38% and ~ 18% respectively in association with improved endurance performance (~ 100% increase in time to exhaustion Burgomaster et al., 2005; and +22% increase in time to exhaustion Shepley et al., (1992) activity post HIT.

#### 3.1.3.1 Variables for adaptation

Similar to improving power, manipulating the intensity of the bout and the work to rest ratio of multiple sprint bouts can stimulate certain aerobic metabolic responses that improve endurance performance (Laursen & Jenkins., 2002). Even short sprint cycle bouts of 10 x 6 sec, (1:10), and 6 x 10 sec sprints, (1:3 (7.5% body mass resistance)) for 6 sessions over a 2 week period have found improvements in time trial and time to exhaustion tests in triathletes and runners (Jakeman, Adamson, Babraj., 2012; Kavaliauskas, Aspe, Babraj., 2015). Indeed, Hazell et al., (2010) have shown that sprint duration does not appear to be important for endurance adaptations, with similar improvements in $VO_2$ max and 5km run time with 30sec (1:8 ratio) and 10sec (1:24 and 1:12 ratios) sprints (Hazell et al., 2010). In addition, Yamagashi & Babraj., (2017) found similar improvements in $VO_2$ peak, time trial, time to exhaustion, and critical power when using 15sec and 30sec sprints but still using a 1:8 work to rest ratio. The reason for this could be the similar metabolic demand of both sprint protocols, resulting in a reduction of PCr and glycogen across the repeated sprints resulting in a shift to aerobic metabolism in later sprints (Hazell et al., 2010; Bogdanis et al., 1995). Lloyd Jones, Morris, Jakeman., (2017) also found similar improvements in self-paced 10km time trial testing (5.1%, 6.2%) after completing 6 HIT sessions when comparing a 6sec sprint and 30sec sprint (both using a 1:8 ratio against 7.5% body mass) when matched for overall sprinting time of 2min. They speculate that both groups may have improved time trial testing due to an increase in citrate synthase activity. The similar increase in citrate synthase in both groups could be due to the shorter rest in the 6sec group and the maintained PPO which may have caused a greater stress on the working skeletal muscles (Lloyd Jones, Morris, Jakeman., 2017). It has also been demonstrated that when matching training groups for work (4 x 30sec sprint 4min rest vs. 2min continuous maximal sprint) there is no

difference in the regulation of cellular energy homeostasis (AMPK phosphorylation (Taylor et al., 2016)). Indicating that short duration exercise (6sec sprint) can have similar endurance adaptations to longer duration exercise (30sec sprint (Lloyd Jones, Morris, Jakeman., 2017; Taylor et al., 2016)).

Another variable that may affect endurance outcomes to HIT is work to rest ratio. Hazell et al., (2010) found a significant improvement in endurance performance regardless of work to rest ratio, with 5km time trial performance improved for all groups and $VO_{2max}$ for 1:24 and 1:8 work to rest ratio groups only. In contrast, Kavaliauskas, Aspe, Babraj., (2015) found that a lower work to rest ratio leads to greater endurance adaptations, with 3km time trial improved only in 1:3 work to ratio group and time to exhaustion improved in the 1:3 and 1:8 but not the 1:12 work to rest ratio group (Kavaliauskas, Aspe, Babraj., 2015).

### 3.1.4 Self-regulated recovery

It has been shown that young adult participants (18-35 years) can SR their recovery time effectively to maintain sprint speed performance (12 x 30m (Glaister et al., 2010)) and MPO (10 x 6 seconds, 7.5% body mass resistance (Phillips, Thompson, Oliver., 2014) to maintain maximal performance (coefficient of variation (CV (≤ 5.2%)). Both Glaister et al., (2010) and Phillips Thompson, Oliver., (2014) have shown that young adults can self-regulate recovery between sprints. However, in Study 1 we found that not all participants managed to do so with 20% of participants failing to do so. Glaister et al., (2010) have suggested that participants with a lower level of aerobic capacity would choose longer rest periods, suggesting the longer the rest period would indicate an increase in fatigue, and this could be used as a surrogate indicator for fatigue. However, Phillips Thompson, Oliver., (2014) suggests that participants could be using pacing strategies during SR rest to prevent any homeostatic disturbances that could lead to early exercise termination (Tucker et al., 2006). It is believed that participants pace their efforts to prioritise energy expenditure (Edwards & Polman., 2013). Pre planning will identify what the task

is, the importance of the task, the person's capabilities and willingness to do the task (Edwards & Polman 2013). Therefore, during a sprint protocol participants will select a longer rest duration if there is no willingness to complete the next sprint, due to the factors above, and select a shorter rest duration if there is a willingness to complete the next sprint.

During self-paced cycling exercise, there is evidence to suggest that the exercise is regulated through sensory feedback to the central nervous system (CNS) through central fatigue (Davis., 1995; Froyd et al 2016; Kay et al 2001; Meeusen et al., 2006; Noakes et al 2001; St Clair Gibson et al 2001; Swart et al 2009; Swart et al 2012; Tucker et al 2006). Central fatigue is defined as a reduction in maximal capacity of the CNS to optimally recruit motor units to produce force (Gandevia., 2001). Central and peripheral fatigue, through afferent feedback (from peripheral organs: lungs, heart and skeletal muscle), work together to ensure that the participant's peripheral critical threshold is never exceeded (Amann., 2011; Amann., 2012; Froyd et al 2016). The peripheral critical threshold is defined as the reduction in the muscle capacity and the reduction in the neuromuscular junction to prevent maximal force (Froyd et al 2016). It is thought that peripheral fatigue is a regulator for self-paced cycling exercise by reducing the amount of muscle recruitment through afferent feedback (Amann., 2011; Amann., 2012; Froyd et al 2016). Afferent feedback comes from sensory nerves located within muscle spindles and Golgi tendon organs (Marcora., 2008; Proske., 2005). These sensory nerves sense tension, position and movement, and then send signals through the CNS to give the sense of effort (Proske., 2005). Both central and peripheral fatigue are believed to contribute to neuromuscular fatigue during exercise (Froyd et al 2016). It has been demonstrated that when intensity during exercise bouts increase there is also an increase in neuromuscular and peripheral fatigue (Amann & Dempsey., 2008). However, these studies have used self-paced time trial cycling and not looked at central or peripheral fatigue during rest periods. What can be highlighted from these studies is that peripheral fatigue occurs 20% into the time trial and steadily increases as the time trial continues (Froyd, Millet, Noakes., 2013). Whereas central fatigue is thought to occur only after peripheral fatigue had already developed (Decorte et al., 2012) and further

develops depending on the exercise duration (Place et al., 2010). Giving the lack of research in central and peripheral fatigue during HIT specifically in recovery periods, it would suggest from the above that peripheral fatigue plays a larger role during exercise and therefore would control duration of SR rest. Therefore, SR rest could be regulated by afferent feedback.

Phillips Thompson, Oliver., (2014) and Study 1 identified that male and female participants over-estimated their SR rest period by at least 10%. There is a potential that pacing tactics may have occurred in both sexes when SR rest is reduced by 15%, due to the significant drop in MPO when compared to the criterion sprint (~ -5.1%) and trial 3 (~ -4.1% (Study 1)). This could potentially explain how the participants were able to keep their CV% to ≤ 5.2%. Pacing tactics may have occurred to prevent homeostatic disturbance that would have led to early exercise failure (Tucker et al., 2006). Using SR rest to repeatedly reproduce similar or greater MPOs of a criterion sprint during training could lead to improvements in endurance and power output.

### 3.1.5 Aims and hypothesis

Therefore, the aim to this study is to determine whether training adaptations are similar between SR and set work to rest ratios. It is hypothesised that SR rest training will lead to greater improvements in power output vs fixed rest (30 sec) training. Given that participants are able to maintain MPO (Phillips Thompson, Oliver., 2014; Study 1). With this maintenance of power, it would suggest that participants would increase their anaerobic pathway stores (PCr and glycogen) and see an improvement in neural responses (Rodas et al., 2000; Creer et al., 2004), therefore improve MPO and PPO. It is also hypothesised that the fixed rest training will lead to greater endurance adaptations. Due to the decrement in performance between sprints, which in turn will allow a greater aerobic presence during and between sprints.

## 3.2 Methods

### 3.2.1 Participants

Twenty-four physically active young males, took part in more than the American College of Sports Medicine and American Heart Association recommended 2.5 hours of moderate physical activity (Haskell., 2007) volunteered for this study. Participants completed 6 ± 2 h structured activity per week consisting of training and playing team sports. All participants were required to be either competitive or recreational athletes, aged 18-35 years and trained > 3 hours per week. Before taking part, participants were given written and verbal instructions about the study prior to giving informed consent. Participants also completed a physical activity readiness questionnaire to ensure there was no known health issues that would put the participants in harm by taking part this study. Participants were stratified into three group based on $VO_2$ peak: SR group (n = 8, 178 ± 7 cm, 78 ± 7 kg, 48 ± 8 $ml.min^{-1}.kg^{-1}$), fixed rest (FR) group (n = 8, 178 ± 4 cm, 81 ± 8 kg, 48 ± 7 $ml.min^{-1}.kg^{-1}$), and control (C) group (n = 8, 180 ± 6 cm, 80 ± 13 kg, 51 ± 9 $ml.min^{-1}.kg^{-1}$). Abertay University ethics committee granted ethical approval for the study and the study was carried out in line with the declaration of Helsinki.

### 3.2.2 Procedures

#### 3.2.2.1 Pre and post testing

At the beginning of the study, participants' body mass (BM, kg) and height (cm) were recorded using a digital scale (Tanita SA 165A-0950U-3) and digital stadiometer (Seca 264), respectively. Participants underwent 3 separate testing sessions, 24 hours apart, pre and post intervention, which involved : $VO_2$ peak, time to exhaustion (TTE), and haemoglobin blood test (test day 1), a single 30 second Wingate test (test day 2), and 10km time trial test (test day 3).

#### 3.2.2.2 Haemoglobin, $VO_2$ peak and time to exhaustion

Haemoglobin concentration, haematocrit % and haemoglobin ratio were measured in pre and post testing (same testing day as $VO_2$ peak test), by using

a haemoglobin photometer (EKF Diagnostics). Haemoglobin ratio was calculated by using the formula as shown in equation 3.1.

Haemoglobin ratio = hen % / hemo.
*Equation 3.1: Where hen % is haematocrit percentage, and hemo is haemoglobin concentration.*

A single blood sample was taken from the participant's index finger prior to the $VO_2$ peak test, the skin was punctured using an Accu-check single use lancet (Roche Diagnostics, UK). Pressure was applied to the finger to draw blood, the first blood sample was wiped away with a sterile cotton wipe, and pressure to the finger was applied again for a second blood sample and placed within a haemoglobin microcuvette (EKF Diagnostic), to collect ~ 8µl of blood. Blood measures were taken from the right hand as this has shown to be more reliable than using the left hand (CV = 6.3%, r = 0.69), and continued to be more reliable after four days of consecutive testing (CV = 7%, r = 0.5) when using a photometer (Morris et al., 1999). After the photometer had analysed the blood sample, haemoglobin concentration was presented as $mmol.L^{-1}$ and haematocrit was presented as a percentage. Using a portable photometer to calculate haemoglobin and haematocrit percentage from capillary blood has been proven to be a reliable measure (Morris et al., 1999). When comparing laboratory measures and using a portable photometer data appears to be very similar (CV = 1%, r = 0.99). Indicating that using a portable photometer for haemoglobin measures is a reliable measure with similar readings to laboratory methods (Morris et al., 1999).

Participants then underwent a cycle $VO_2$ peak test. Participants were fitted with a mask connected to an online expired gas analyser (Cortex Metalyzer 3B). Participants then mounted the cycle ergometer (Monark Ergomedic 894), which was adjusted to ensure full leg extension, and cycled for 4 minutes at 60 W. Immediately following this warm up, power output increased by 30 $W \cdot min^{-1}$ until volitional exhaustion or participants could no longer maintain 60 RPM (Jakeman, Adamson, Babraj., 2012). Participants were instructed to maintain a

cadence of 60 RPM and were verbally encouraged throughout. $VO_2$ peak was determined as the highest 30 sec average across the test (See section 3.2.2.2).

The duration of the $VO_2$ peak test was recorded (sec) and was defined as a participant's TTE (Jakeman, Adamson, Babraj., 2012; Stevens & Dascombe., 2015), which is another measure of endurance capacity (Stevens & Dascombe., 2015). This will allow a measure of how long a participant can perform at a specific speed (60 RPM) whilst cycling at a steady state increasing intensity (30 watt increase each minute), and allows a comparison of physiological markers ($VO_2$ peak for the purpose of this thesis) between testing periods (Laursen et al., 2007). An increase in TTE is a reflection of an increase in power output (Hopkins, Schabort, Hawley., 2001), which is suggested to be an indicator of improving endurance performance (Bulbulian, Wilcox, Darbos., 1986; Noakes., 1988).

#### 3.2.2.3 Wingate (30 sec sprint) test

Seat height was adjusted on the ergometer (Monark Ergomedic 894E, Sweden) to ensure the participant had a full leg extension, prior to the test. The participants then cycled for 60 seconds at 60rpm against a resistance of 1kg. Participants then completed a 30 second all out Wingate sprint. Once the participant reached 110rpm the resistance was added and the sprint began. Strong verbal encouragement was given throughout. After the sprint PPO was recorded as the highest $W.kg^{-1}$ value, and MPO was recorded as the overall $W.kg^{-1}$ mean average of the sprint.

Within study 2 a single 30 sec Wingate test was used to measure maximal anaerobic power and anaerobic capacity (Vandewalle, Pérès, Monod., 1987) before and after the intervention. Similar to using multiple Wingate sprints for HIT (Burgomaster et al., 2005, 2006, 2008; Hazell et al., 2010; Lloyd Jones, Morris, Jakeman., 2017; Yamagishi & Babraj., 2017), 7.5% of the participant's body mass is typically used for a Wingate test (Vandewalle, Pérès, Monod., 1987). The purpose of the Wingate test within study 2 was to measure the maximal watt value (maximal anaerobic power) known as PPO (typically

achieved within the first 5-10 sec of the sprint) and measure the total amount of work (overall average watt value) known as MPO which is an index of anaerobic capacity (Vandewalle, Pérès, Monod., 1987). PPO will be a reflection of the predominate use of PCr (48%) and glycolysis (43%). MPO is then therefore a reflection of ATP turnover from 10-30 sec, which is predominately oxidative phosphorylation (> 50%), with glycolysis (~35%) and PCr (~15%) making up for the rest of ATP turnover (Parolin et al., 1999). Using a Wingate test to measure anaerobic capacity has proven to be a reliable measure, it is highly correlated between test-retest by r = 0.91 (Ayalon, Inbar, Bar-or., 1974) and r = 0.93 (Patton, Murphy, Fredrick., 1985).

#### 3.2.2.4 Time trial and time trial rational

Again, the seat height was adjusted on the ergometer to ensure the participant had a full leg extension, prior to the test. The participants then cycled for 4 min at 60 revolutions per minute (RPM) against 1kg of resistance. Participants then completed a 10km cycle on the ergometer, as fast as possible. The rpm was self-selected by the participant as they cycled against a fixed resistance of 2kg (60W) (Jakeman, Adamson, Babraj., 2012). Participants were not informed of time during the time trial until post testing was complete. Distance completed was the only visible feedback participants received.

To assess endurance performance time trial (TT) tests are commonly used by using self-selected pace with the objective to cover a specific distance as quickly as possible (Burgomaster et al., 2005, 2006, 2008; Hazell et al., 2010; Kavaliauskas, Aspe, Babraj., 2015; Lloyd Jones, Morris, Jakeman., 2017; Stevens & Dascombe., 2015; Yamagishi & Babraj., 2017). HIT research has found improvements in TT performance by 5-10% (Burgomaster et al., 2005, 2006, 2008; Hazell et al., 2010; Kavaliauskas, Aspe, Babraj., 2015; Lloyd Jones, Morris, Jakeman., 2017; Stevens & Dascombe., 2015; Yamagishi & Babraj., 2017). TT testing has been shown to be a reliable test-retest measure (CV = 3.4%, r = > 0.95) for predicting competitive endurance performance (Dantas, Pereira, Nakamura., 2015; Jeukendrug et al., 1996).

In studies 2 and 3, a 10km cycle (Monark peak bike 894) TT was used to asses endurance performance. Prior to this participants completed a warm up consisted of 4 min cycling at 60 rpm against 1kg as resistance on a cycle ergometer. Participants were then instructed to complete a self-paced 10km cycle time trial as quickly as possible with a fixed resistance (males – 2kg, females – 1.5kg). Only distance covered was reported to the participants. In study 3 females used a lighter resistance than males due to morphological differences in muscle and fat mass between sexes, which results in a greater power output in males compared to females (Perez-Gomez et al., 2008). Using a fixed resistance would replicate a realistic time trial performance in the sense that if participants were to perform a 10km time trial cycle on a track/course they would all have to cycle against the same gradient. However, with the overall aim of a time trial to maintain a high speed for a long period of time, participants that can produce greater amounts of force will cycle faster (Bulbulian, Wilcox, Darbos., 1986; Noakes., 1988). Therefore, morphological differences between participants would give a greater advantage to participants that have a greater muscle mass and or greater body mass (Perez-Gomez., 2008). Using percentage of body mass as a resistance for a time trial may allow the test to be fairer and allow participants to produce similar power outputs (Billaut & Bishop., 2009). However, using a fixed resistance during a time trial (males: 2kg, females: 1.5kg) is commonly used (Jakeman, Adamson, Babraj., 2012; Kavaliauskas, Aspe, Babraj., 2016; Yamagishi & Babraj., 2017).

#### 3.2.2.5 Sprint training warm up

In all training sessions, participants were required to complete a warm up that consisted of 4 min cycling at 60 RPM against 1kg of resistance on a cycle ergometer. Once completed a sprint specific warm up was carried out; consisting of 3 x 3 second sprints against 7.5% body mass with an active 45s recovery consisting of cycling at 50-60 RPM (with no resistance) between sprints. Participants then rested for 4 minutes prior to completing the sprint trials (Phillips Thompson, Oliver., 2014). After resting, participants could perform any light stretches that they wished.

#### 3.2.2.6 Criterion sprint

In trial 1 participants underwent the sprint training warm up procedure before completing the criterion sprint (CS) test. The CS test consisted of a single 6 sec cycle sprint against a resistance equal to 7.5% body mass to familiarise them with the procedure and to provide criterion sprint data (MPO $W.kg^{-1}$) for comparison with repeated sprint performance. Once the 6 sec sprint was completed, participants cycled at 60rpm for 1 minute against a resistance of 1kg. Participants then came off the ergometer and rested for 5 minutes before repeating another single 6 sec sprint. If participants achieve a lower MPO in test 2, the result of test 1 was taken as the participants MPO (Phillips Thompson, Oliver., 2014). If participants achieved a MPO in test 2 that is ≥ 5% greater than test 1, a third test was undertaken (Phillips Thompson, Oliver., 2014). This was repeated as necessary until MPO no longer increases (Phillips Thompson, Oliver., 2014; see section 3.2.3.3 for further details on the CS).

#### 3.2.2.7 Sprint training

10 x 6 second sprints (see section 2.2.3.2 for sprint duration rational) were completed in each training session with either a FR or SR rest between sprints, against 7.5% body mass resistance (see section 2.2.3.1 for body mass resistance rational). Resistance was applied to the flywheel once participants reached 110 RPM. The aim for the SR group was to take as much time as required during the active recovery (cycling against no resistance at 50-60 RPM) to enable them to perform a maximal effort in all sprints (instructions were adapted from Glaister et al 2010; and Phillips Thompson, Oliver., 2014; see section 2.2.3.4 for SR instructions). The aim for the FR group was to perform maximally across all 10 sprints after 30 seconds of active/passive recovery. Participants were informed to remain seated during each sprint and no external feedback was given to the participants apart from cadence. During each sprint participants were verbally encouraged to cycle to a maximal effort. The rest period was defined as the moment from the bike cradle weight being lifted until it dropped again to start the next sprint. Participants were not informed on the duration of their rest period. During training sessions 1 and 6 participants had

heart rate recorded throughout (Bioharness; see section 2.2.5.2 for further details on recording heart rate).

### 3.2.3 Statistical analysis

Statistical analysis for the study was analysed on IBM SPSS Version 22.0 software. Two-way (group x test) repeated mixed linear measures analysis of variance (ANOVA) compared magnitude of change for haemoglobin and haematocrit %, $VO_{2peak}$, time to exhaustion, PPO, MPO, and time trial. A further two-way repeated mixed linear (group x trial) ANOVA compared mean normalised heart rate in training sessions 1 and 6, for rests 1 vs. 9 and sprints 1 vs. 10. Bonferroni post hoc analysis explored significant main effects. Significant main effects between groups were further explored by using an independent samples T test. If sphericity was violated then Greenhouse-Geisser corrections were used. Cohen's D was used to measure effect size between the SR rest group and the fixed rest group. Effect size was defined as trivial (0.0-0.2), small (0.2-0.5), moderate (0.6-1.1), and large (1.2-1.9 (Cohen., 1992)). Negative effect size values indicate a greater change in the fixed rest group and positive effect size values indicate a greater change in the SR group. Magnitude of change between pre and post testing was calculated as post value – pre value / pre value *100. Statistical significance was as accepted at $p \leq 0.05$, data are mean with ± standard deviation (SD), and figures 2.1-2.3 data are magnitude of change with ± standard error measurement (SEM). SEM was employed instead of standard deviation in magnitude of change measurements, due to the larger variability. Using standard error of measurement allowed a true reflection of the magnitude in change error by considering group size. A Pearson's correlation was used to identify a link between maintenance of MPO data, haemoglobin measures, sprint HR, and resting HR between percentage change of performance measures. A Pearson's correlation was also used to identify a link between the percentage change of performance measures from pre to post against other performance measures (example: correlation between TTE and TT).

## 3.3 Results

### 3.3.1 Self-regulated recovery duration

Mean results for self-regulated recovery duration is shown in Table 3.1. In the SR group there was no significant main effect between trials for rest duration ($F_{1,5} = 1.037$, $p > 0.05$).

### 3.3.2 Mean power output

A main effect was present between trials for MPO ($F_{2.933,1} = 8.639$, $p < 0.05$), post hoc indicates that the CS is significantly greater than trial 1 (Table 3.1). A main effect was also present between groups for MPO ($F_{1,14} = 9.668$, $p < 0.05$), post hoc indicates that MPO is significantly higher in trials 1-6 for the SR group compared to FR group ($p < 0.05$). No significant interaction was present ($F_{2.933,1} = 2.38$, $p > 0.05$).

### 3.3.3 Mean power output percentage change

A significant main effect was found in MPO % change between the CS and trials 1-6 ($F_{2.443,1} = 3.436$, $p < 0.05$), post hoc is unable to identify where significance lies (Table 3.1). A significant main effect between groups is also present ($F_{1,13} = 5.366$, $p < 0.05$), post hoc indicates that FR MPO % change is significantly greater than the SR group in when comparing trial 1 and 4 to the CS. No significant interaction was present ($F_{2.443,1} = 0.426$, $p > 0.05$).

### 3.3.4 Fatigue index

FI% found no main effect between trials ($F_{1,5} = 0.624$, $p > 0.05$; Table 3.1), and no interaction ($F_{1,5} = 0.688$, $p > 0.05$). However, a main effect between groups for FI% data was present ($F_{1,14} = 69.274$, $p < 0.05$), post hoc indicates that FR group FI% data is significantly higher across all trials compared to SR group data ($p < 0.05$).

### 3.3.5 Coefficient variation

A significant main effect was found between trials for CV % ($F_{5,1} = 4.519$, $p < 0.05$; Table 3.1), post hoc indicates that CV % in trial 1 is significantly higher vs. trials 2/3/6 ($p < 0.05$). A significant main effect was also found between groups ($F_{1,14} = 8.412$, $p < 0.05$), FR group CV % is significantly greater than SR group in all trials ($p < 0.05$). No significant interaction was present for CV % data ($F_{5,1} = 0.896$, $p < 0.05$).

Table 3.1: Performance variables across the 6 trials of self-regulated and fixed rest (30sec) repeated sprint exercise for both groups.

| Measures | Criterion Sprint | Trial 1 | Trial 2 | Trial 3 | Trial 4 | Trial 5 | Trial 6 |
|---|---|---|---|---|---|---|---|
| **Recovery time (sec)** | | | | | | | |
| SR | - | 84 ± 30 | 114 ± 63 | 108 ± 35 | 113 ± 46 | 101 ± 39 | 105 ± 44 |
| **MPO (W.kg $^{-1}$)** | | | | | | | |
| SR | 11.29 ± 1.6[a] | 10.48 ± 1.52* | 10.98 ± 1.63* | 10.76 ± 1.54* | 11.03 ± 1.7* | 11.09 ± 1.44* | 11.10 ± 1.54* |
| FR | 10.57 ± 1.06[a] | 8.86 ± 0.84 | 9.14 ± 0.78 | 9.34 ± 0.69 | 9.27 ± 0.75 | 9.24 ± 1.06 | 9.44 ± 0.98 |
| **MPO (W.kg $^{-1}$) % change (CS vs. trial)** | | | | | | | |
| SR | - | -7.1 ± 3.3 | -2.8 ± 3.4 | -4.5 ± 4.6 | -2.2 ± 4.9 | -1.7 ± 4.3 | -1.4 ± 5.4 |
| FR | - | -15.9 ± 7.8[b] | -12.7 ± 12.2 | -10.9 ± 11.6 | -11.6 ± 10.2[b] | -11.9 ± 13.5 | -10 ± 11.9 |
| **Fatigue index (%)** | | | | | | | |
| SR | - | 7.29 ± 3.06 | 3.57 ± 1.6 | 4.52 ± 2.36 | 6.26 ± 4.58 | 5.88 ± 2.34 | 6.64 ± 2.97 |
| FR | - | 12.31 ± 3.48‡ | 11.92 ± 3.93‡ | 12.45 ± 3.82‡ | 11.28 ± 4.47‡ | 11.77 ± 4.46‡ | 12.08 ± 4.58‡ |
| **CV %** | | | | | | | |
| SR | - | 5.74 ± 3.22 | 2.81 ± 0.91† | 3.42 ± 2.22† | 4.48 ± 2.78 | 3.99 ± 2.24 | 3.87 ± 1.16† |
| FR | - | 10.85 ± 5.56‡ | 9.03 ± 4.16‡† | 9.56 ± 5.69‡† | 9.2 ± 5.28‡ | 9.38 ± 6.08‡ | 8.17 ± 3.41‡† |

*Data shown as mean average ± standard deviation. * Significantly greater than corresponding FR data. ‡ Significant greater than corresponding SR data. † Significantly lower than trial 1. [a] Significantly greater than trial 1. [b] Significantly greater than corresponding SR data.*

### 3.3.6 Percentage change for endurance measures

#### 3.3.6.1 $VO_2$ peak

Percentage change for $VO_2$ peak ($ml.kg^{-1}.min^{-1}$), time to exhaustion (TTE) (seconds), and 10km TT (seconds) for pre to post percentage change testing results for all groups are shown in figure 3.1. In $VO_2$ peak, no significant difference was present between groups in pre testing ($F_{2, 23} = 0.411$, $p > 0.05$; SR: 47.8 ± 7.5; FR: 48 ± 7.1; C: 50.1 ± 8.6 $ml.kg^{-1}.min^{-1}$). No significant main effect was present in percentage change post testing between all groups but there was a medium effect between groups ($F_{2, 23} = 0.995$, $p > 0.05$; SR vs. FR: $d = -0.7$).

#### 3.3.6.2 Time to exhaustion

In TTE, no significant difference was present between groups in pre testing ($F_{2, 23} = 1.275$, $p > 0.05$; SR: 544 ± 75; FR: 601 ± 42; C: 586 ± 96sec). No significant main effect was present in percentage change post testing between all groups but there was a large effect between groups ($F_{2, 23} = 0.27$, $p > 0.05$; SR vs. FR: $d = 0.8$).

#### 3.3.6.3 Time trial

In TT, no significant difference was present between groups in pre testing ($F_{2, 22} = 1.427$, $p > 0.05$; SR: 1013 ± 175; FR: 895 ± 50; C: 992 ± 185sec). No significant main effect was present in percentage change post testing between all groups but there was a large effect between groups ($F_{2, 22} = 1.51$, $p > 0.05$; SR vs. FR: $d = -1.1$).

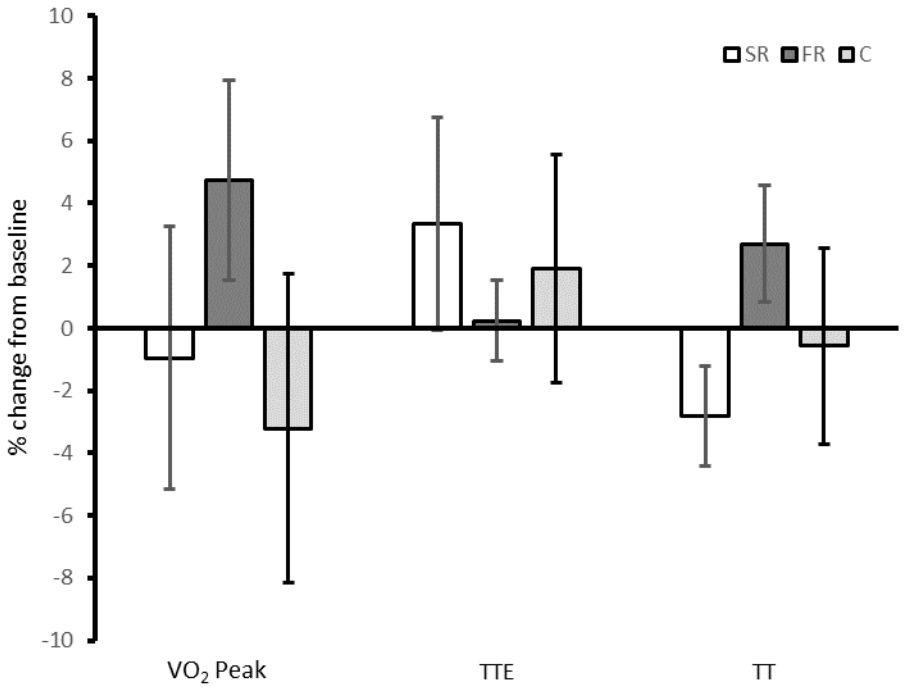


*Figure 3.1: $VO_2$ peak, TTE and TT percentage change in SR, FR and C groups from pre to post testing.*

### 3.3.7 Percentage change for power measures

#### 3.3.7.1 Wingate peak power output

Power measures during a 30 sec Wingate test for PPO ($W.kg^{-1}$), and MPO ($W.kg^{-1}$) for pre to post percentage change testing results for all groups are shown in figure 3.2. In PPO, no significant main effect was present between groups in pre testing ($F_{2, 22} = 0.151$, $p > 0.05$; SR: 11.9 ± 2.1; FR: 12.4 ± 2.4; C: 11.8 ± 2.1 $W.kg^{-1}$). No significant main effect was present in percentage change post testing between all groups but there was a medium effect between groups ($F_{2, 22} = 0.342$, $p > 0.05$; SR vs. FR: d = 0.5).

#### 3.3.7.2 Wingate mean power output

In MPO, no significant difference was present between groups in pre testing ($F_{2, 22} = 0.37$, $p > 0.05$; SR: 8 ± 1.1; FR: 8.1 ± 0.6; C: 8.1 ± 1.1 $W.kg^{-1}$). No significant main effect was present in percentage change post testing between

all groups, with a small effect between groups ($F_{2, 22} = 0.441$, $p > 0.05$; SR vs. FR: d = 0.3).

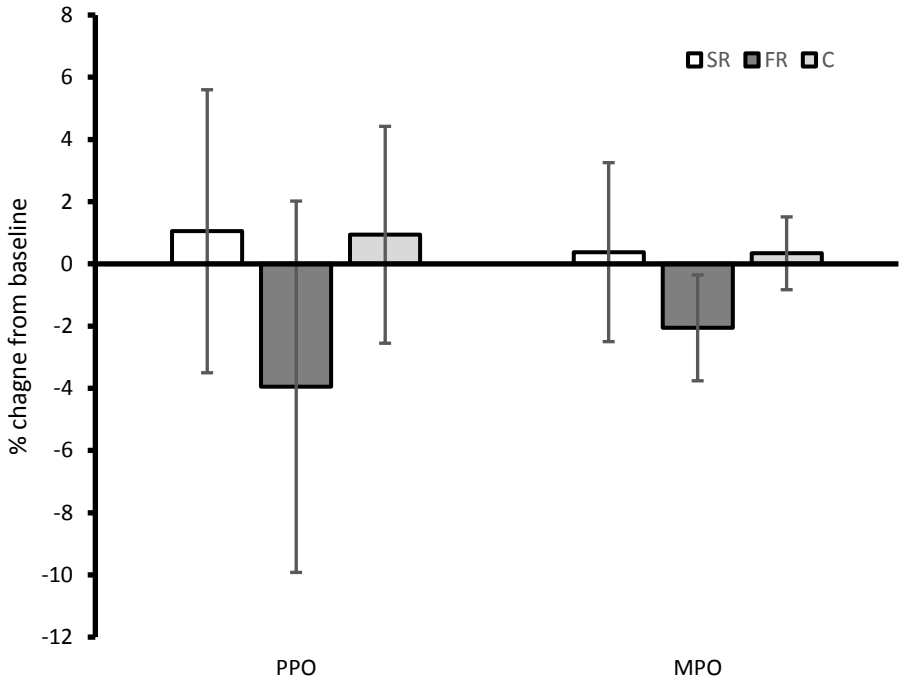


*Figure 3.2: Wingate PPO and MPO percentage change in SR, FR and C groups from pre to post testing.*

### 3.3.8 Performance measures correlations

Table 3.2 shows correlation values (r) comparing between the percentage change of in session MPO and the CS (MPO% of CS), in session CV% and FI% for the overall percentage changes of performance tests whilst combining both training groups' data. No significant correlations occurred in MPO% of CS and: $VO_2$ peak ($r = -0.44$, $p > 0.05$) TTE ($r = 0.09$, $p > 0.05$), TT ($r = -0.14$, $p > 0.05$), PPO ($r = -0.2$, $p > 0.05$), MPO ($r = -0.01$, $p > 0.05$). No significant correlations occurred in CV% and: $VO_2$ peak ($r = 0.38$, $p > 0.05$) TTE ($r = 0.02$, $p > 0.05$), TT ($r = 0.17$, $p > 0.05$), PPO ($r = 0.27$, $p > 0.05$), MPO ($r = -0.09$, $p > 0.05$). No significant correlations occurred in FI% and: $VO_2$ peak ($r = 0.29$, $p > 0.05$) TTE ($r = 0.04$, $p > 0.05$), TT ($r = 0.32$, $p > 0.05$), PPO ($r = -0.18$, $p > 0.05$), MPO ($r = -0.28$, $p > 0.05$).

Table 3.2: correlations values between percentage change of in session MPO against the CS, in session CV and FI for performance measures percentage changes.

| Measure | $VO_2$ peak | TTE | TT | PPO | MPO |
|---|---|---|---|---|---|
| **MPO% of CS** | r = -0.44 | r = 0.09 | r = -0.14 | r = -0.2 | r = -0.01 |
| **CV%** | r = 0.38 | r = 0.02 | r = 0.17 | r = 0.27 | r = -0.09 |
| **FI%** | r = 0.29 | r = -0.04 | r = 0.32 | r = -0.18 | r = -0.28 |

*Correlation values comparing MPO% of CS, CV% and FI% between percentage change in performance tests.*

Table 3.3 shows correlation values (r) for the overall percentage changes of performance tests whilst combining both training groups' data. A significant correlation occurred within Wingate PPO and MPO ($r = 0.61$, $p < 0.05$).

Table 3.3: correlations values between percentage change between performance measures.

| Test | $VO_2$ peak | TTE | TT | PPO | MPO |
|---|---|---|---|---|---|
| **$VO_2$ peak** | - | r = -0.13 | r = 0.18 | r = 0.23 | r = 0.26 |
| **TTE** | r = -0.13 | - | r = -0.41 | r = 0.05 | r = 0.03 |
| **TT** | r = 0.18 | r = -0.41 | - | r = -0.26 | r = -0.31 |
| **PPO** | r = 0.23 | r = 0.05 | r = -0.26 | - | r = 0.61* |
| **MPO** | r = 0.26 | r = 0.03 | r = -0.31 | r = 0.61* | - |

*Correlations of combined training groups and sexes percentage change in performance tests.* ***** *Significant positive correlation ($p < 0.05$).*

### 3.3.9 Haemoglobin measures

#### 3.3.9.1 Haemoglobin

Haemoglobin (mmo/l), haematocrit % and haemoglobin ratio measures for pre to post percentage change testing results for all groups are shown in figure 3.3. In haemoglobin, no significant main effect was present between groups in pre testing ($F_{2, 23} = 0.764$, $p > 0.05$; SR: 8.8 ± 0.8; FR: 8.8 ± 0.8; C: 9.2 ± 0.5 mmo/l). No significant main effect was present pre vs. post testing between all groups but there was a medium effect between groups ($F_{2, 23} = 0.404$, $p > 0.05$; SR vs. FR $d = 0.5$).

#### 3.3.9.2 Haematocrit

In haematocrit % no significant main effect was present between groups in pre testing ($F_{2, 22} = 0.689$, $p > 0.05$; SR: 41.9 ± 3.8; FR: 42.6 ± 3.4; C: 43.8 ± 2.4 %). No significant main effect was present in percentage change post testing between all groups and there was a small effect between groups ($F_{2, 22} = 0.721$, $p > 0.05$) SR vs. FR: $d = 0.3$).

#### 3.3.9.3 Haemoglobin ratio

In haemoglobin ratio no significant main effect was present between groups in pre testing ($F_{2, 22} = 0.948$, $p > 0.05$; SR: 4.7 ± 0.04; FR: 4.7 ± 0.03; C: 4.7 ± 0.03). No significant main effect was present in percentage change post testing between all groups but there was a medium effect between groups ($F_{2, 22} = 2.143$, $p > 0.05$; SR vs. FR: $d = -0.5$).

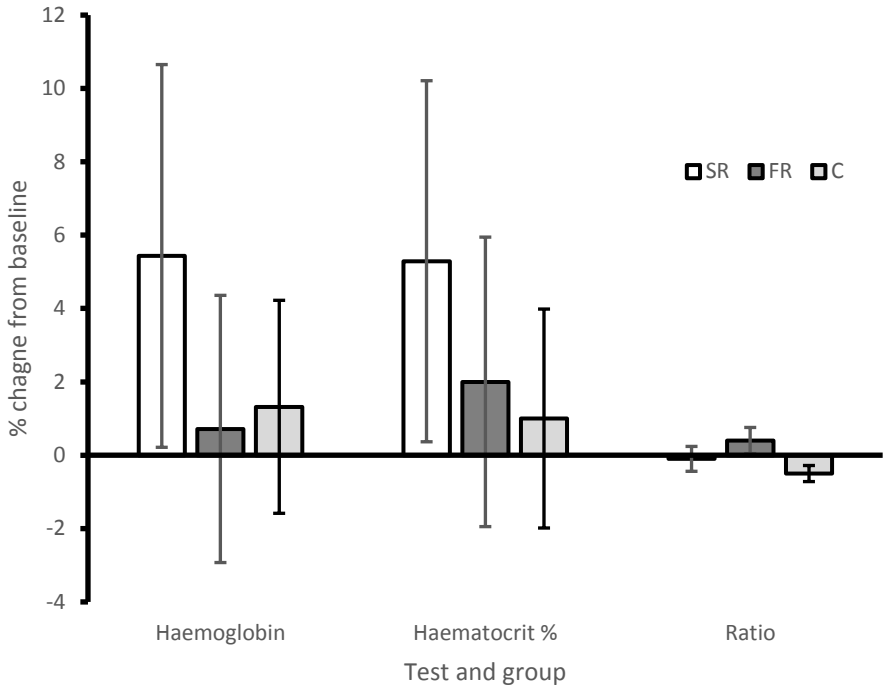


*Figure 3.3: Haemoglobin, haemoglobin haematocrit % and haemoglobin ratio percentage change in SR, FR and C groups from pre to post testing.*

Table 3.4 shows correlation of haemoglobin measures between percentage changes of performance measures. A significant correlation occurred within haemoglobin and TTE ($r = 0.73$, $p < 0.05$), haematocrit% and TTE ($r = 0.69$, $p < 0.05$), and ratio and TTE ($r = -0.58$, $p < 0.05$).

Table 3.4: correlation between haemoglobin measures and performance measures.

| Test | $VO_2$ peak | TTE | TT | PPO | MPO |
|---|---|---|---|---|---|
| **Haemoglobin** | r = -0.37 | r = 0.73* | r = -0.29 | r = 0.35 | r = 0.07 |
| **Haematocrit%** | r = -0.35 | r = 0.69* | r = -0.26 | r = 0.38 | r = 0.02 |
| **Ratio** | r = -0.12 | r = -0.58** | r = 0.14 | r = -0.02 | r = 0.01 |

*Correlation values comparing percentage change of haemoglobin, haematocrit and ratio between percentage change in performance tests.* ***** *Significant positive correlation (p < 0.05). ** Significant negative correlation (p < 0.05).*

### 3.3.10 Normalised sprint heart rate data

Figure 3.4 shows heart rate (HR) for sprint 1 (S1) and sprint 10 (S10) for trials 1 and 6 for SR and FR groups, A: shows normalised sprinting HR between the two groups, B: shows normalised trial 1 S1 and S10 HR curve, C: shows normalised trial 6 sprint S1 and sprint S10 HR curve. In normalised sprinting HR, a significant main effect was present between trials and sprint periods ($F_{3, 38.845} = 23.259$, $p < 0.05$), and between groups ($F_{1, 13.867} = 3.273$, $p < 0.05$). Post hoc indicates that all sprint 10 data is significantly greater than all sprint 1 data ($p < 0.05$). No significant main effect was present between groups ($F_{1, 13.867} = 3.273$, $p < 0.05$). No significant interaction effect was present ($F_{3, 38.845} = 1.921$, $p > 0.05$).

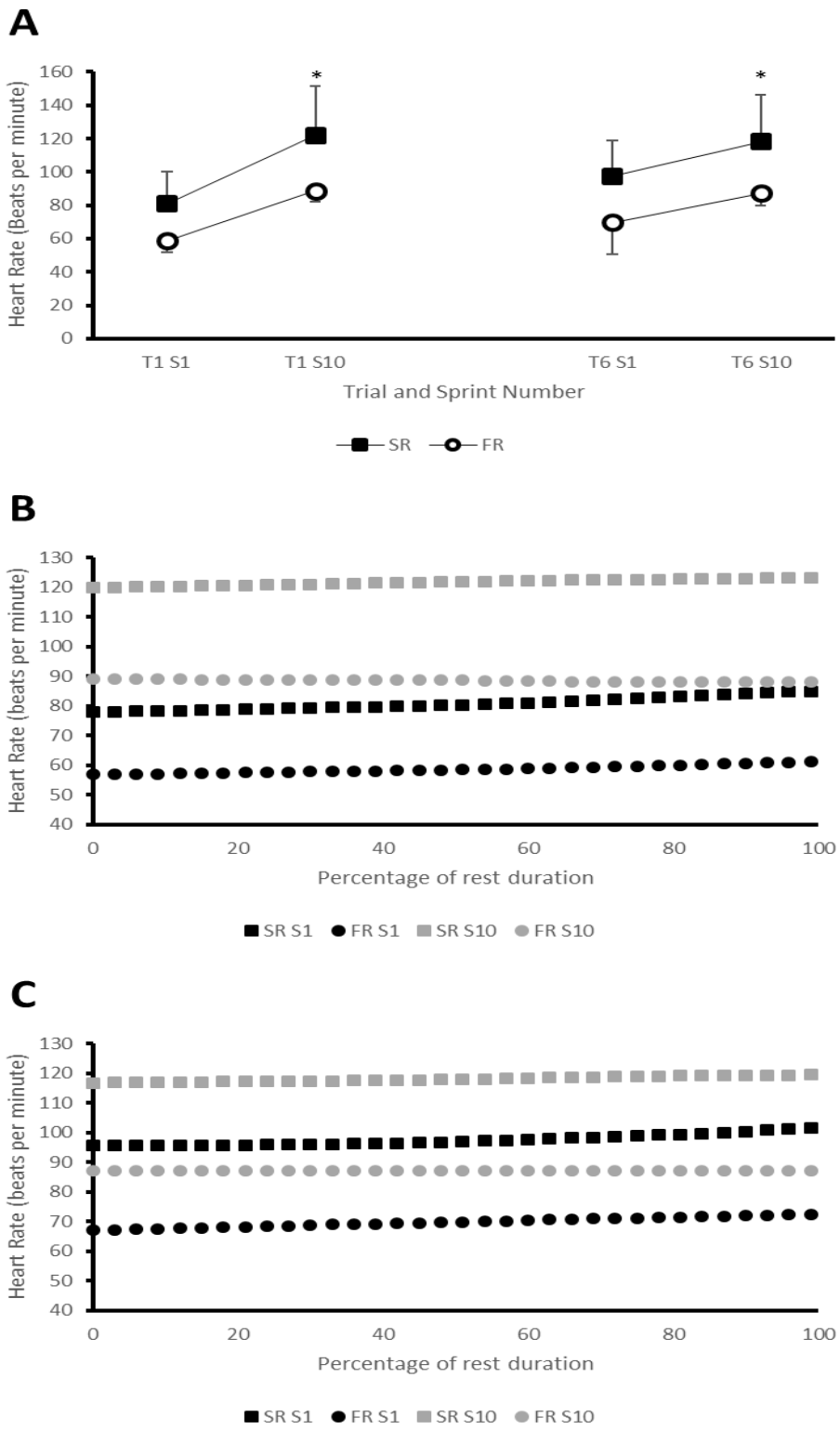


*Figure 3.4: A: Normalised average sprinting HR sprint 1 and sprint 10 between trials 1 and 6 for SR and FR groups. * Significantly greater than all sprint 1 data. B: Normalised sprinting HR curve data showing sprints 1 and 9 in trial 1. C: Normalised sprinting HR curve data showing sprints 1 and 9 in trial 6 for SR and FR groups.*

### 3.3.11 Normalised resting heart rate data

Figure 3.5 shows heart rate (HR) for rest 1 (R1) and rest 9 (R9) for trials 1 and 6 for SR and FR groups, A: shows normalised resting HR between the two groups, B: shows normalised trial 1 R1 and R9 HR curve, C: shows normalised trial 6 R1 and R9 HR curve. In normalised resting HR, a significant main effect was present between trials and rest periods ($F_{3, 39.591} = 5.239$, $p < 0.05$), and between groups ($F_{1, 15.156} = 10.545$, $p < 0.05$). Post hoc indicates that trial 1 R9 is significantly greater than trial 1 R1 ($p < 0.05$). Post hoc between groups indicates that SR group HR is significantly greater than FR group in trial 1 R1, R9 and trial 6 R9 ($p < 0.05$). No significant interaction effect was present ($F_{1, 39.591} = 1.017$, $p > 0.05$).

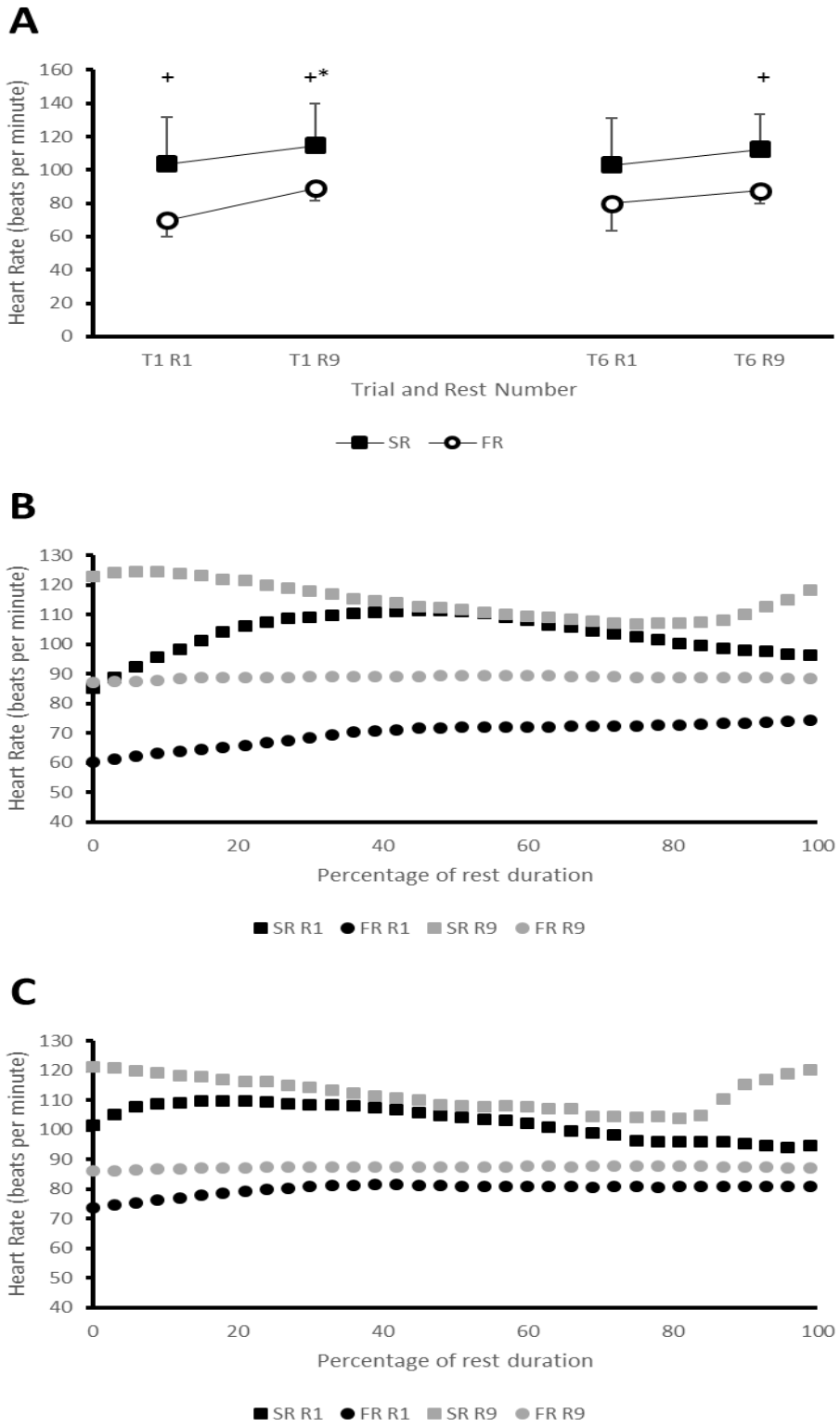


*Figure 3.5: A: Normalised average resting HR rest 1 and rest 9 between trials 1 and 6 for SR and FR groups. * Significantly greater than trial 1 rest 1. + Significantly greater than FR group data. B: Normalised resting HR curve data showing rests 1 and 9 in trial 1. C: Normalised resting HR curve data showing rests 1 and 9 in trial 6 for SR and FR groups.*

### 3.3.12 Haemoglobin correlations

Table 3.5 shows correlation of normalised sprint and resting HR between percentage changes of performance measures. Normalised sprint and resting HR measures are overall total of trials 1 and 6 rests 1 and 9 data (TOTAL).

Table 3.5: correlation between sprint and resting HR, and percentage change of performance measures.

| Measure | $VO_2$ peak | TTE | TT | PPO | MPO |
|---|---|---|---|---|---|
| **HR:** | | | | | |
| Sprint TOTAL | r = -0.62† | r = 0.23 | r = -0.18 | r = 0.05 | r = -0.56 |
| Rest TOTAL | r = -0.51 | r = 0.22 | r = -0.21 | r = 0.03 | r = -0.47 |

*Correlation values comparing TOTAL in sprint and resting normalised HR between percentage change in performance tests. Data is from trials 1 and 6 rests 1 and 9. † Approaching significant negative correlation (p = 0.056).*

## 3.4 Discussion

The aim to this study was to determine whether training adaptations were similar between SR and fixed work to rest ratios. It was hypothesised that SR rest training would lead to greater improvements in power output vs. FR group, and that the FR group would lead to greater endurance adaptations vs. SR group. Interestingly there is no significant difference in either group in $VO_2$ peak, TTE, TT, PPO, MPO, haemoglobin, haematocrit %, and haemoglobin ratio pre vs post testing ($p > 0.05$). Percentage change within the performance tests and effect size (Cohen's D) does show small, moderate and large effect sizes between the two training groups. With the SR group experiencing a larger percentage increase in TTE, TT, PPO and MPO compared to the FR group. The FR group shows a greater percentage change in $VO_2$ peak compared to the SR group. It was also hypothesised that maintaining MPO would lead to improvements in PPO and MPO during a 30 sec Wingate test. However, correlation data (Table 3.2) indicates that maintenance of MPO, CV% and

lowered FI% is not essential for increasing power output or any of the endurance tests.

### 3.4.1 Trial data

Table 3.1 data suggests that the SR group were able to maintain CV% of MPO in trials 2-6 (trial 1 CV% is > 5.2%) and did so significantly greater than FR group in all trials ($p < 0.05$). Trials 2, 3 and 6 CV% data was also significantly less than trial 1 for both groups ($p < 0.05$). FI% was also significantly less in the SR group when compared to the FR group across all trials ($p < 0.05$). Therefore, the CV% and FI% data would suggest that the SR group were successful in maintaining MPO across trials 2-6, and that the 30 sec FR between sprints was effective in creating a decrement in MPO performance. However, the percentage change from the CS vs. MPO within trials 1-6 suggests that the SR group may have adopted pacing tactics, similar to the findings of female MPO data in Study 1. It has previously been demonstrated that pacing occurs during a single bout of exercise (Wittekind, Micklewright, Beneke., 2011). Wittekind, Micklewright, Beneke., (2011) found that PPO was significantly greater in a 5 sec sprint compared to a 15, 30 and 45 sec sprint despite PPO usually been achieved between 0-5 seconds (Vandewalle, Pérès, Monod., 1987). SR participants may have identified that seeking to maintain their CS MPO over 10 x 6 sec sprints was too challenging and adapted their efforts (Wittekind, Micklewright, Beneke., 2011). Further explaining why CV% and FI% is unaffected and similar between trials 2-6. Therefore, SR participants did not keep within the aim of the study and did not maintain their maximal effort repeatedly. As expected, due to a shorter work:rest ratio, the FR group experience a greater percentage change in MPO from the CS and trials when compared to the FR group, with a significance appearing in trials 1 and 4 (FR trial 1: ~ -15.9%, SR trial ~ -7.1%, FR trial 4: ~ -11.6%, SR trial 4: ~ -2.2%, $p < 0.05$). A significant main effect between trials is present for both groups, with the CS sprint MPO been significantly greater than trial 1 MPO. Both groups appear to have increased their average MPO as the number of trials increased but did not do so significantly. It is thought that FR group experienced a greater aerobic demand due to the shorter work to rest ratio compared to the SR group

(FR: 1:5. SR: ~1:17 (Gaitanos et al., 1993; Kavaliauskas, Aspe, Babraj., 2015)). The greater aerobic demand during the sprints and rest periods could indicate that that there isn't sufficient time to fully recover PCr with a shortened fixed rest would lead to a decrement in MPO (Gaitanos et al., 1993; Kavaliauskas, Aspe, Babraj., 2015). This is evident from Table 3.1 power, CV% and FI% data.

Correlation data that compares Table 3.1 measures against percentage increase in performance testing (Table 3.2) indicates that maintenance of power during HIT is not related to improving performance in endurance or power output. Data in Table 3.2 is not significant; however, correlation values (r) suggest that improving $VO_2$ peak is related to varying MPO (seeking a decrement in MPO) within trials for both training groups. This is suggested by the negative correlation (r = -0.44) of percentage change between the CS and trial 1-6 MPO and $VO_2$ peak. A greater CV% and FI% percentage also seems to have a link in improving $VO_2$ peak (CV% r = 0.38, FI% r = 0.29). Suggesting that varying MPO during HIT with a shorter work to rest ratio increases aerobic activity during HIT (Gaitanos et al., 1993; Gosselin et al., 2012), which is consistent to an improvement in $VO_2$ peak (Kavaliauskas, Aspe, Babraj., 2015). There is also the possibility that decreasing FI% (maintaining MPO) is a factor for improving 10km TT performance (r = 0.32) and MPO (r = -0.28). HIT research is consistent with finding improvements in a greater sustained work rate or PPO and MPO (Creer et al., 2004; Forbes, Slade, Meyer., 2008; Hazell et al., 2010; Jakeman, Adamson, Babraj., 2012; Kavaliauskas, Aspe, Babraj., 2015; Kavaliauskas, Steer, Babraj., 2016; Lloyd Jones, Morris, Jakeman., 2017; Ørtenblad et al., 2000; Rodas et al., 2000). Participants that improved their TT performance may have done so by greater motor unit activation, increase $Ca^{2+}$ release from the sarcoplasmic reticulum, improved $MTC_{1,4}$ activity, increased glycogen availability, and lactate metabolism, which are all linked to improving TT performance post HIT (Burgomaster et al., 2006; Creer et al 2004; Jakeman, Adamson, Babraj., 2012; Juel et al., 2003; Ørtenblad et al., 2000). Increasing glycogen availability and increasing $Ca^{2+}$ release and uptake within the sarcoplasmic reticulum would decrease the amount of Pi, which is in keeping with maintaining power output (Ørtenblad et al., 2000; Westerblad, Allen, Lannergren., 2002). Given that the TT in the present study was against a fixed

resistance, an increase in power output would allow participants to pedal faster and therefore decrease their TT. These potential adaptations post HIT may explain why the SR group improved their TT performance despite a negative change in $VO_2$ peak. Why the FR group saw a negative response to TTE and TT despite an increase in $VO_2$ peak is surprising given that increasing $VO_2$ max is correlated to increase TTE, due to an increase in PPO (r = 0.86), and increasing MPO is correlated to increasing TT performance (r = 0.93 (Driller., 2012)). This could indicate that increasing TTE and TT performance is greatly associated with anaerobic measures (as discussed above), potentially explaining why HIT research has found increases in TTE and TT despite no change in $VO_2$ peak or max (Burgomaster et al., 2005, 2006; Kavaliauskas, Aspe, Babraj., 2015; Kavaliauskas, Steer, Babraj., 2016; Lloyd Jones, Morris, Jakeman., 2017).

### 3.4.2 Performance measures

#### 3.4.2.1 $VO_2$ peak

$VO_2$ peak (SR: -1 ± 4.2%, d = -0.2. FR: 5 ± 3.2%, d = 1.5. C: -3 ± 4.9%, d = -0.7 (Figure: 3.1)) saw no significant changes between pre and post testing in all groups ($p > 0.05$). It was hypothesised that the FR group would improve greater than the SR group due to a shorter work to rest ratio (FR: 1:5, SR: ~1:17) that would create a greater aerobic response during HIT (Gaitanos et al., 1993; Gosselin et al., 2012; Kavaliauskas, Aspe, Babraj., 2015). However, HIT research is not consistent with increasing $VO_2$ peak or $VO_2$ max (Burgomaster et al., 2005; Kavaliasukas, Steer, Babraj., 2016; Linossier et al., 1993; Lloyd Jones, Morris, Jakeman., 2017), possibly explaining the no change in $VO_2$ peak for the SR group. Kavaliasukas, Steer, Babraj., (2016) discuss that the no change in $VO_2$ peak for their participants could be due to the short duration of the study (2 weeks). Astorino & Schubert., (2014) found a greater response in participants increasing $VO_2$ max (78% of participants) when using a long term HIT protocol (12 weeks) when compared to participants (65%) using a short term protocol (2 weeks). Therefore, literature would suggest that the SR group

within the present study may experience an increase in $VO_2$ peak if the duration of the present HIT protocol increased by > 2 weeks.

Hazell et al., (2010) speculates that the reproducibility of power output may be a key factor for improving endurance power measures. However, in the present study non-significant correlation data in Table 3.2 indicates that not maintaining CS MPO (r = -0.44), varying power output by a decrement in force (CV r = 0.38), and causing greater fatigue (FI r = 0.29) is linked with increasing $VO_2$ peak. The decrement in force in relation to the CS and during the trial indicates a dominant shift in energy metabolism from PCr and glycolysis to aerobic metabolism (Bogdanis et al., 1996; Gaitanos et al., 1993; Kavaliasukas, Aspe, Babraj., 2015). Kavaliasukas, Aspe, Babraj., (2015) speculate that increasing aerobic demand and decreasing power output during HIT may be a key factor for improving endurance measures. Therefore, within the present study participants may have increased their $VO_2$ peak by using a rest duration that reduces ATP turnover due to a depletion in PCr and inhibition of anaerobic glycolysis (Bogdanis et al., 1996; Gaitanos et al., 1993).

Increasing haemoglobin (r = -0.37), haematocrit (r = -0.35) and haemoglobin ratio (r = -0.12) is not significantly correlated to increasing $VO_2$ peak (Table 3.4). The SR group experience the greatest increase in haemoglobin (SR: 5 ± 5.2%, d = 1, p > 0.05. FR: 1 ± 3.6%, d = 0.2, p > 0.05. C: 1 ± 2.9%, d = 0.5, p > 0.05) and haematocrit (SR: 5 ± 4.9%, d = 1.1, p > 0.05. FR: 2 ± 3.9%, d = 0.5, p > 0.05. C: 1 ± 3%, d = 0.3, p > 0.05 (Figure 3.3)) yet experienced a negative magnitude in change in $VO_2$ peak. Given that haemoglobin is a contributing factor in the delivery of oxygen (Pottgiesser, Schumacher., 2013; Warburton et al., 2000), it would suggest that an increase in oxygen kinetics increased, which would explain why there is no change in $VO_2$ peak for the SR group but increases in all other performance measures (Kavaliasukas, Steer, Babraj., 2016; Marsh & Martin., 1997). Menz et al., (2015) found no significant increase in $VO_2$ max and haemoglobin mass after 11 HIT sessions with trained athletes. Menz et al., (2015) explains that several studies using trained athletes experience no link between $VO_2$ max and haemoglobin after HIT. The increase in $VO_2$ peak for the FR group could due to an increase in citrate synthase

activity, which is linked to an increase in $VO_2$ max following HIT (Vigelso, Andersen, Dela., 2014). Citrate synthase activity may have increased due to a greater aerobic demand in the FR group due to the shorter work:rest ratio, given its adaptability following aerobic training (Vigelso, Andersen, Dela., 2014).

Reducing sprint and recovery TOTAL normalised HR (Table 3.5) is potentially linked to increasing $VO_2$ peak respectively ($r = -0.62$, $p < 0.075$; $r = -0.51$). Both training groups sprint and recovery HR significantly increase from sprint 1/ rest 1 vs. sprint 10/ rest 9 within trials 1 and 6 (Figure 3.4, 3.5). However, rest 9 HR data is significantly greater in the SR group vs. the FR group in trials 1 and 6. This suggest that increasing the aerobic demand during HIT or a decrement in trial MPO leads to a decreased HR due to a steady decline in power output. Yamagishi & Babraj., (2017) found similar normalised HR measures along with similar lactate measures despite using 15 and 30 sec sprints. They also found similar reproducibility of PPO during the HIT sessions, indicating that the SR group experienced a greater use of anaerobic glycolysis during the present study HIT due to a higher normalised HR during sprints and rests. The potential greater use in glycolysis for the SR group and greater aerobic demand for the FR group may explain why a decrease in HR led to an increase in $VO_2$ peak for the FR group and no change for the SR group (Gaitanos et al., 1993). This is in contrast with the work of Kavaliasukas, Aspe, Babraj., (2015) who found a greater HR during HIT with a shorter recovery period. However, Kavaliasukas, Aspe, Babraj., (2015) did not normalise their HR data, which may explain the contrast in findings within the present study.

#### 3.4.2.2 Time to exhaustion

The SR group experienced larger improvements in TTE (3 ± 3.4%, d = 1) when compared to the FR (TTE: 0.2 ± 1.3%, d = 0.2) and a moderate effect size (SR vs. FR d = 0.8). It was hypothesised that the SR group would experience a smaller improvement for endurance based testing when compared to the FR group. Due to longer recovery period, which leads to a smaller aerobic demand during HIT (Gosselin et al., 2012; Kavaliasukas, Aspe, Babraj., 2015). In contrast to this study, Kavaliauskas, Aspe, Babraj., (2015) found significant

increases in TTE with 30 sec (1:3) rest (~ 6.4%, d = 0.8, p = 0.003) and 80 sec (1:8) rest (~ 4.4%, d = 0.6, p = 0.03) whereas 120 sec (1:12) rest saw no significant improvement (~ 1.9%). Using a work:rest ratio of 1:8 appears to be consistent with increasing TTE by 100% (Burgomaster et al., 2005), 12% (Kavaliauskas, Steer, Babraj., 2016), 6.2% and 12.8% (Yamagishi, Babraj., 2017). Further studies have found an increase in TTE (4%) using a work:rest ratio of 1:10 (Jakeman, Adamson, Babraj., 2012), and by 3.5% and 3% when using a work:rest ratio of 1:24 and 1:12 respectively (Hazell et al., 2010). Indicating that shorter rest durations or smaller work:rest ratios are not essential for increasing endurance capacity. This potentially explains why the SR group increased their TTE despite using an average work:rest ratio of ~1:17.

Hazell et al., (2010) speculates that the maintenance of power output may be a key factor for improving performance measures given that using a 30 and 10 sec sprint had similar magnitude in change from pre to post testing and due to a greater maintenance of power using a 10 sec sprint. However, in the present study maintaining CS power output, reducing CV% and FI% of MPO during the trials was not strongly correlated to increasing TTE ($r = 0.09$; $r = 0.02$; $r = -0.04$ respectively). TOTAL HR during sprints ($r = 0.23$, $p < 0.05$) and rest periods ($r = 0.22$, $p < 0.05$) also appears to not be strongly linked to increasing TTE. It is thought that the increasing haemoglobin (SR: ~5%. FR: ~1%) and haematocrit (SR: ~5%. FR: ~2%), and no change in haemoglobin ratio (SR: ~-0.1%. FR: ~0.4%) is linked with improving TTE. This is suggested by the significant correlation between TTE and haemoglobin ($r = 0.73$, $p < 0.05$), haematocrit ($r = 0.69$, $p < 0.05$), and haemoglobin ratio ($r = -0.58$, $p < 0.05$). Increasing haemoglobin concentration would increase the delivery of oxygen to the working muscles during the TTE (Pottgiesser, Schumacher., 2013; Warburton et al., 2000) and has been found to increase aerobic power output (Kanstrup & Ekblom., 1984), which may explain the greater increase in TTE for the SR group compared to the FR group. Haemoglobin concentration may have increased more in the SR group than the FR group due to a greater work:rest ratio, using a shorter work:rest ratio is thought to increase the aerobic demand during HIT (Gosselin et al., 2012; Kavaliasukas, Aspe, Babraj., 2015). It is unsure why the SR group had a greater increase in haemoglobin compared to

the FR group, the differences in recovery duration and or greater maintenance of CS MPO may have caused a greater lowering in the amount of $O_2$ within the blood, which would stimulate erythropoiesis (Mairbaurl., 2013). This releases stem cells from bone marrow that have the ability to self-duplicate and after a maturation process haemoglobin is synthesised with developing erythroblasts (Adamson & Finch., 1975).

Improving TTE could be due to increased muscle citrate synthase activity after HIT, which would increase mitochondrial activity (Burgomaster et al., 2005), and increased resting PCr stores (Burgomaster et al., 2005). Increasing resting PCr stores decreases an onset of fatiguing mechanisms such as lowering pH (~ 0.5 units), build up in Pi and a reduction in impeded $Ca^{2+}$ dynamics (Balsom et al., 1992; MacLaren & Morton., 2012; Westerblad, Allen, Lannergren., 2002). However, increasing PCr and citrate synthase activity is strongly linked (Kent-Braun & Alexander., 2000) and positively linked (Vigelso, Andersen, Dela., 2014) to an increase in $VO_2$ max respectively. Indicating that SR group participants did not increase their TTE due to an increase in PCr or increased citrate synthase activity, given that the SR group saw no increase in $VO_2$ peak. SR group participants may have increased their TTE due to an increase in power output, which would allow a higher sustainability of work rate (Vanhatalo, Doust, Burnley., 2008). This higher sustainability of work rate could be due to an increase in glycogen stores and glycogen availability, due to an increase in the glycolytic flux mechanism, which has been found to increase power output following HIT (7 x 15-30 sec sprints (Rodas et al., 2000)). This is suggested by the ~10% increase in TTE observed in Rodas et al., (2000) following HIT. Research from Jakeman, Adamson, Babraj., (2012) found a significant rightward shift in the lactate curve which resulted in an increase of power (~30W) following 6 sessions of 10 x 6 sec cycle sprints. The SR group may have increased their TTE by changes in lactate metabolism, which would lead to a greater power output during the test. Post HIT has also shown to increase lactate transporter activity $MTC_1$ and $MTC_4$ (Bishop et al., 2008; Burgomaster et al., 2005; Perry et al., 2008). It is thought that $MTC_1$ and $MTC_4$ activity may have increased in the current study due to the substantial rest duration, which would allow time for lactate removal after each sprint (Bishop et al., 2008;

Sahlin et al., 1976). Indicating that the SR group may have increased their TTE by an increase in lactate metabolism, via increased $MTC_1$ and or $MTC_4$ activity, which would lead to a greater power maintenance during the test.

#### 3.4.2.3 Time trial

Similar to the TTE test, the SR group saw a greater improvement in 10km TT testing by decreasing their time by ~-3% whereas the FR increased their time by ~3%. These changes were not significant, however, there was a large effect size when comparing between the SR and FR groups (d = -1.1). Kavaliauskas, Aspe, Babraj., (2015) also found a significant improvement in 3km TT, only in the 30 sec rest group (30 sec rest ~ -3.1% $p < 0.05$; 80 sec rest ~ -2.4% $p > 0.05$; 120 sec rest ~ 2.4% $p > 0.05$). Burgomaster et al., (2005 (~-10%)) Hazell et al., (2010 (1:8: ~-5.2%, 1:24: ~-3.5%, 1:12: ~-3%)), Jakeman, Adamson, Babraj., (2012 (~-10%)), Lloyd Jones, Morris, Jakeman., (2017 (6 sec sprint: ~-5.1%, 30 sec sprint: ~-6.2%)), and Yamagishi, Babraj., (2017 (15 sec sprint: ~-8.6%, 30 sec sprint: ~-17.2%)) also saw significant decreases in TT testing. The present study data appears to be inconsistent with the findings of Kavaliauskas, Aspe, Babraj., (2015), who found greater improvements in 3km TT performance with the shortest work:rest ratio (1:3) when compared to greater work:rest ratios (1:8 and 1:12). They speculate that reducing the work:rest ratio will increase the aerobic demand of the HIT and lead to greater improvements in endurance testing (Gaitanos et al., 1993; Gosselin et al., 2012). In contrast with Jakeman, Adamson, Babraj., (2012), they used double the amount of rest (60 sec) between sprints compared to the FR group in this study and both studies used 10 x 6 sec sprints, yet Jakeman, Adamson, Babraj., (2012) found a decrease of ~10% whereas the FR group increased their time by ~3% . This could be due to a larger maintenance in power across each trial, made possible by 60 sec rest period in comparison to the 30 sec rest period used for this study (Hazell et al., 2010).

Similar to the TTE test, TT performance may have improved due to an increase in lactate metabolism, which is potentially linked to an increased work rate of 30W (Jakeman, Adamson, Babraj., 2012), and or an increase in power output

which potentially increased TTE by ~10% (Rodas et al., 2000). This is potentially indicated by a small correlation link between reducing FI% and TT (r = 0.32 (Table 3.2)). As FI% increases a decrement in power also increases (Bogdanis., 2012), when this occurs glycogen stores decrease and the aerobic metabolism becomes the dominant fuel source for the sprints (Pette., 1985). Suggesting that the SR group were able to continue using glycogen as an energy source for their sprints more than the FR group (Pette., 1985). Small and moderate correlations between TT, increasing Wingate PPO (r = -0.26), increasing Wingate MPO (r = -0.31), and increasing TTE (r = -0.41) could suggest that the increases in all these tests for the SR group are linked (Table 3.3). Jakeman, Adamson, Babraj., (2012) explain a decrease in blood lactate could be due to an increase in skeletal muscle uptake of lactate, due to an increase in $MTC_{1,4}$ activity (Burgomaster et al., 2007). There is also the possibility that that the SR group improved their TT performance due to an increase in greater glycogen stores (Rodas et al., 2000), which would increase power output and allow participants to sustain a higher work rate (Vanhatalo, Doust, Burnley., 2008). Therefore, SR participants may have improved their TT performance by been able to pedal faster against the same fixed resistance from the pre TT test.

#### 3.4.2.4 Wingate power output testing

No significant difference between testing and groups was found in PPO (SR: 1 ± 4.5%, $p > 0.05$. FR: -4 ± 6%, $p > 0.05$. C: 1 ± 3.5%, $p > 0.05$) and MPO (SR: 0.4 ± 2.9%, $p > 0.05$. FR: -2 ± 1.7%, $p > 0.05$. C: 0.3 ± 1.2%, $p > 0.05$ (Figure 3.2)). When comparing effect size between the SR and FR groups, there is a moderate effect size in PPO (d = 0.5) and small effect size in MPO (d = 0.3). Further indicating a greater increase in PPO and MPO in the SR group. This is consistent with the work of Kavaliauskas, Aspe, Babraj., (2015) who found that longer work:rest ratios (1:8 and 1:12) found greater improvements in Wingate PPO (~8.5% and ~7.1%) and MPO (~4.6% and ~5.3%) following HIT compared to a shorter work:rest ratio (1:3, ~4.3% and ~0.3%). From their study it was hypothesised that the SR group would see larger improvements in PPO and MPO due to a higher training power output average. Previous HIT research has

shown to increase power output due to an improvement in neurological responses (firing rate), remodelling of muscle fibres, increased PCr stores or recovery, glycogen stores, and improved $Ca^{2+}$ dynamics (Burgomaster et al., 2005; Creer et al., 2004; Ørtenblad et al., 2000; Pette., 1985; Rodas et al., 2000). The repetition of producing multiple maximal efforts would lead to a dominant recruitment of type II fibres and use in glycolytic enzyme activity (Roepstorff et al 2006; Russ et al., 2005). In comparison, the FR group saw a drop in both PPO and MPO, which may explain the lack of positive change in the TTE and TT tests despite an increase in $VO_2$ peak. Hazell et al (2010) found a significant increase in PPO and MPO when using 10 and 30 sec sprints with 4min recovery, however, they only found a significant increase in PPO when using 10 sec sprints with 2min recovery (MPO $p = 0.06$). Further suggesting that a longer recovery during HIT is a factor for improving PPO and MPO.

A moderate correlation was present in TOTAL normalised sprinting HR and increasing MPO ($r = -0.56$ (Table 3.5)). A similar correlation occurred in TOTAL normalised resting HR and increasing MPO ($r = -0.47$). Potentially suggesting that increasing HR during sprints and during rest periods is important for improving MPO. Given that normalised HR was higher in the SR group than the FR group in sprints but also significantly higher during trial 1 rest 1 and rest 9, and trial 6 rest 9. There is also a significant correlation between MPO and PPO ($r = 0.61$, $p < 0.05$ (Table 3.3)). These correlations suggest that seeking to increase HR during HIT leads to an improvement to PPO and MPO (Table 3.5). PPO and MPO may not have increased as much as recent research (Hazell et al., 2010; Kavaliauskas, Aspe, Babraj., 2016) due to the known 10% over-estimation in required SR recovery (Phillips Thompson, Oliver., 2014; Study 1).Phillips Thompson, Oliver., (2014) discusses that this 10% over-estimation in SR may not stimulate the correct physiological responses to improve performance testing. Further research should identify if PPO and MPO are increased when SR is reduced by > 10%.

### 3.4.3 Limitations

The current study did not take into account what sport, activity or competition phases the participants take part in. This may have caused tiredness or lethargic states for some of the participants who were midway through their competition calendar. The potential tiredness or lethargic states could have affected TTE, TT, Wingate PPO and MPO in post testing for the FR group, given that previous similar research (10 x 6 sec cycle sprints, 60 sec rest between sprints) has found a ~10% improvement in TT and ~4% improvement in TTE (Jakeman, Adamson, Babraj., 2012). The difference of 30 sec in rest between the FR group in the current study and Jakeman, Adamson, Babraj., (2012) would previously indicated towards a greater improvement in TTE and TT due to a potentially greater aerobic demand during the HIT because of the shorter rest period (Kavaliauskas, Aspe, Babraj., 2015).

In the current study it is thought that the SR group experienced a greater normalised aerobic demand during the HIT trials compared to FR group. Given that normalised HR was greater in the SR group, Study 1 shows a significant increase in $VO_2$ and $VCO_2$ measures from rest 1 to rest 9, and Study 1 also shows an association between greater sum of trial MPO, $VO_2$ and $VCO_2$. However, this is speculative and gas masks within trials, to measure $VO_2$ and $VCO_2$, were not used due to potential discomfort factors discussed in Study 1 (see section 3.5). Using gas masks to measure and normalise $VO_2$ and $VCO_2$ between the two training groups could further indicate that seeking to maintain CS MPO ten times in a single trial leads to a greater aerobic demand compared to using a shorter work:rest ratio, as used by the FR group.

### 3.4.4 Conclusion

This study demonstrates that maintaining power through SR rest leads to greater improvements in TTE, TT, Wingate PPO and MPO when compared to varying power output. Increasing haemoglobin concentration, haematocrit, and HR measures during HIT appear to be key factors for improving TTE, TT, Wingate PPO and MPO, which was achieved more greatly when using SR rest compared to a 30 sec FR period. Seeking to maintain CS MPO during HIT is

not strongly correlated to increasing any of the performance measures. However, seeking to cause a decrement in MPO during HIT appears to be moderately correlated to increasing $VO_2$ peak, possibly due to an increasing aerobic demand during HIT (Kavaliauskas, Aspe, Babraj., 2015). This present study is also consistent with previous literature that found smaller work to rest ratios lead to greater increase in $VO_2$ peak and greater work to rest ratios lead to greater power outputs (Kavaliauskas, Aspe, Babraj., 2015). Data from the SR group may not be significant due to the established 10% overestimated SR rest (Phillips Thompson, Oliver., 2014; Study 1). Further research should demonstrate the effects of HIT and SR rest but aim to remove the 10% overestimated rest during HIT.

### 3.4.5 Practical implications

The present study has identified four major findings. 1) Created further evidence that HIT over a two-week period (6 sessions) using short duration sprints (10 x 6 sec sprints against 7.5% body mass resistance) leads to increased performance in endurance and power measures. 2) The use of SR rest leads to greater improvements in TTE, TT and Wingate power output measures compared to a FR, which may create tailored rest periods for each participant. 3) Using a FR between sprints and perhaps creating a greater aerobic demand during the HIT creates greater improvements in $VO_2$ peak. 4) A participant's ability to maintain their CS MPO during HIT whilst using SR rest may be affected by the instructions or their interpretation of the instructions given by the researcher. Males been able to maintain their CS MPO during HIT more greatly in Study 1 (Table 2.1) compared to Study 2 (Table 3.1) suggests this. Practitioners should consider what performance outcomes they desire from their athletes when contemplating using SR recovery, and consider their word choice when explaining what they want their athletes to achieve when using SR recovery. Practitioners should also treat the current data as populations specific until further research has been conducted with elite athletes.

### 3.4.6 Proceeding research

The present study found that the SR group experience greater improvements in TTE, TT, Wingate power output and haemoglobin measures compared to the FR group. Whereas the FR experience greater improvements in $VO_2$ peak compared to the SR group. However, this study has not identified if the over-estimation in SR rest (Phillips, Thompson, Oliver., 2014; Study 1) has a negative impact on magnitude in change for the endurance and power output measures. There is also an uncertainty as to what endurance adaptations would occur in female participants, given that they cannot maintain their MPO as greatly as males when using SR rest (Study 1). Therefore, the proceeding research will seek to remove this over-estimation in SR rest during a HIT intervention, and compare HIT effects between males and females.

# 4 Chapter 4 – Study 3 (Sex differences and changes in endurance measures using reduced self-regulated recovery during high intensity training)

## 4.1 Introduction

### 4.1.1 Adaptations to high intensity training

A number of different High intensity training (HIT) protocols have been utilised with differences in work to rest ratio, sprint duration and number of sprints (Burgomaster et al., 2005, 2006, 2008; Hazell et al., 2010; Kavaliauskas, Steer, Babraj., 2015; Kavaliauskas, Steer, Babraj., 2016; Lloyd Jones, Morris, Jakeman., 2017; Rodas et al., 2000; Yamagishi & Babraj., 2017). When total sprint work is balanced (20 x 6 sec sprints 4 x 30 sec sprints) or total number of sprints is the same (4-6 x 15 sec sprints compared to 4-6 x 30 sec sprints) and work to rest ratio is the same then the reported endurance adaptations (time to exhaustion, time trial, critical power and $VO_2$ peak) are similar regardless of sprint duration (Lloyd Jones, Morris, Jakeman., 2017; Yamagishi & Babraj., 2017). This may reflect that glycogen depletion is a major regulator of endurance adaptation (Bogdanis et al., 1996, 1998; Parolin et al 1999) to HIT and if enough sprints are performed then the extent of glycogen depletion is identical regardless of sprint duration (Bogdanis et al., 1998). The majority of phosphocreatine (PCr) and glycogen degradation occurs within the first 15 sec of a 30 sec sprint (Bogdanis et al., 1996, 1998; Parolin et al 1999), suggesting that the last 15 sec of a 30 sec sprint is not vital for improving endurance. When Yamagishi & Babraj., (2017) compared two HIT duration protocols, they found using half the amount of time during HIT led to similar training adaptations in $VO_2$ peak, time to exhaustion (TTE), time trial (TT) and critical power (CP). Research also indicates that as long as there are multiple sprint bouts during HIT there will be a decrease in anaerobic dominance and an increase in aerobic dominance during HIT when using 6-30 sec long sprints and separated by 30 sec to 4 min recovery (Bogdanis et al., 1996, 1998; Parolin et al 1999; Gaitanos et al., 1993). There is an increase in research that is showing that improvements in endurance ($VO_2$ max, $VO_2$ peak, TT and TTE) are able to occur when using HIT protocols that use 6-10 sec long sprints (Hazell et al.,

2010; Jakeman, Adamson, Babraj., 2012; Kavaliauskas, Aspe, Babraj., 2015; Lloyd Jones, Morris, Jakeman., 2017). Shorter sprint periods (5-10sec) have found muscle metabolite, such as PCr and glycogen, increases compared to using a 30sec sprint (Burgomaster et al., 2005 and 2006; Forbes, Slade, Meyer., 2008; Linnosier et al 1993; Ørtenblad et al., 2000; Rodas et al., 2000). Protocols using 30sec sprints saw increases in glycogen by ~26%, ~50%, 32.3% (Burgomaster et al., 2005, 2006; Rodas et al., 2000), and PCr has previously increased by ~31%, ~3.7% and ~1% (Burgomaster et al., 2005, 2006; Rodas et al., 2000). Glycogen and PCr stores have also recorded an increase after performing a 30sec sprint by ~57.9% and ~48% respectively (Rodas et al., 2000). During exercise (60% and 90% of $VO_2$ peak) has seen increases in glycogen and PCr following HIT by > 50% and ~13%, > 100% and ~33% respectively (Burgomaster et al., 2006). Duration of PCr recovery, following high intensity leg extension exercise, has also been found to decrease (~14%) following six sessions of HIT (4-6 x 30sec sprints 4min rest against 7.5% for males and 6.5% for females body mass (Forbes, Slade, Meyer., 2008)). Protocols using 5-10sec sprints have also seen increases in glycogen by ~11.5% (Ørtenblad et al., 2011), glucose 6 phosphate by ~59.1% and PCr by ~5.3% (Linnosier et al., 1993). However, increasing PCr after HIT is not consisted as Ørtenblad et al., (2000) found a decrease in resting PCr (~ 10%) following five weeks of HIT (10sec sprints). After repeat running sprint training (over 8 weeks: 2 x 30sec sprints 10min rest twice a week, 6-10 x 6sec sprints 54sec rest once a week, 5 x 2min run 5min rest once a week) PCr also saw a decrease at rest (~ 1%) and after a 30sec sprint (~ 8.9% (Nevill et al., 1988)).

The use of 30 sec sprint protocols increasing endurance capacity is linked to increasing $VO_2$ peak, citrate synthase activity, glycogen stores and phosphocreatine (PCr) stores (Burgomaster et al., 2005; Burgomaster et al., 2006; Burgomaster et al., 2008; Kent-Braun & Alexander., 2000; Rodas et al., 2000). Increasing citrate synthase activity is a reflection of an increase in mitochondrial activity, which is an important adaptation in endurance training to improve capacity and performance (Holloszy & Coyle., 1984). Increased glycogen and PCr stores following HIT (Burgomaster et al., 2005; Burgomaster et al., 2006; Burgomaster et al., 2008; Ørtenblad et al., 2011; Rodas et al.,

2000) also increase sarcoplasmic reticulum calcium ($Ca^{2+}$) release, which is in keeping with a greater force development (Ørtenblad et al., 2011; Westerblad, Allen, Lannergren., 2002). Increasing glycogen (through an increase glycolytic enzyme activity phosphofructokinase, lactate dehydrogenase (Rodas et al., 2000)) and PCr stores is advantageous for seeking to maintain a high rate of muscular contraction for a sustained period of time (Gaitanos et al., 1993; Ørtenblad et al., 2011; Rodas et al., 2000; Westerblad, Allen, Lannergren., 2002).

During multiple sprint bouts, using 10 x 6sec sprints with 30sec recovery (1:5 work:rest ratio), degradation of PCr is accounted for 49.6% of ATP resynthesis in sprint 1, whereas in sprint 10 PCr accounts to 80.1% of ATP resynthesis (Gaitanos et al., 1993). Therefore, sprint 10 power output was supported by PCr degradation and an increase in aerobic metabolism (Gaitanos et al., 1993). The hydrolysis of ATP produces ADP and inorganic phosphates (Pi) when producing maximal bursts of muscular contraction (Glaister., 2005). During PCr hydrolysis, lactic acid is ionized and produces lactate and $H^+$ (Glaister., 2005). Accumulation of Pi are thought to be causes for peripheral fatigue, which prevents PCr resynthesis and affects calcium ($Ca^{2+}$) dynamics (Glaister., 2005; Ørtenblad et al., 2011; Westerblad, Allen, Lannergren., 2002). This release in Pi leads to an impairment in $Ca^{2+}$ release (muscular contraction) and absorption (muscular relaxation) within the sarcoplasmic reticulum (Ørtenblad et al., 2011; Pilegaard et al., 1999; Westerblad, Allen, Lannergren., 2002). This impairment in $Ca^{2+}$ kinetics directly impacts force production in the myofibrils and reduces sarcoplasmic reticulum $Ca^{2+}$ ATPase (SERCA) concentration (Periasamy & Kalyanasundaram., 2007). A reduction in SERCA leads to a decrement in $Ca^{2+}$ been pumped back (absorbed) to the sarcoplasmic reticulum (Periasamy & Kalyanasundaram., 2007).

Anaerobic capacity and skeletal muscle power is regarded as a contributing factor for improving endurance performance (Bulbulian et al., 1986; Noakes., 1988). Increasing activity in factors such as lactate monocarboxylate transporters (MTC), specifically $MTC_1$ and $MTC_4$, are strongly linked with increasing endurance testing, mean and peak power output testing (Pilegaard

et al., 1999). Increased MTC activity leads to a larger blood flow (~16%) due to an increase in lactate concentration and lactate uptake in skeletal muscle tissue (Gladden., 2000). As lactate increases 75-80% of the lactate is oxidised with the remaining 25-20% been converted into glucose and glycogen (Brooks., 2000). Therefore, allowing a greater ATP turnover to allow higher muscular contraction rates (Bogdanis et al., 1996; Gaitanos et al., 1993). Increasing resting muscle glycogen content, post HIT, has previously been demonstrated (Burgomaster et al., 2005, 2006, 2008). It is possible that an increase in glycogen is responsible for cellular $Ca^{2+}$ homeostasis driving the sarcoplasmic reticulum $Ca^{2+}$ pump, which will cause functional coupling of ATP caused by sarcoplasmic reticulum glycolytic enzymes, allowing a sustaining high rate of muscular contraction (Ørtenblad et al., 2011; Pilegaard et al., 1999; Westerblad, Allen, Lannergren., 2002). Increasing muscular power is regarded as an important factor for improving endurance performance such as a 1.5-10km time trial (Bulbulian et al., 1986; Noakes., 1988). A greater resynthesis of ATP through glycolysis will allow participants to maintain a higher speed during important stages of the time trial such as the start and finish (Bulbulian et al., 1986; Joyner & Coyle., 2008).

Increasing muscular power is due to a greater neuromuscular function, increased $Ca^{2+}$ dynamics and muscle fibre distribution (Allemeier et al., 1994; Bell et al., 2015; Creer et al., 2004; Esbjörnsson et al., 1993; Jacobs et al., 1987; Jansson et al., 1990; Ørtenblad et al., 2000; Yu, Carlsson, Thornell., 2004). HIT (4-6 30sec sprints 4min recovery against 7.5% body mass,15 x 10sec sprints with 50sec recovery against 7% body mass, 2-6 x 15 and 30sec sprints against 7.5% body mass, 4-6 x 30sec sprints 15-20min recovery against 75g per kg body mass), has previously demonstrated a change in dominance in muscle fibre recruitment during exercise (Allemeier et al., 1994; Esbjörnsson et al., 1993; Jacobs et al., 1987; Jansson et al., 1990). Alterations have shown to decrease the recruitment of faster type muscle fibres (type IIX) and increase the recruitment of intermediate fast twitch muscle fibres (type IIA (Allemeier et al., 1994; Esbjörnsson et al., 1993; Jacobs et al., 1987; Jansson et al., 1990; Pette., 1998; Pette & Staron., 1997; Ross & Leveritt., 2001)). This also leads to a decrease in type I muscle fibre recruitment and in some cases shows no alteration in the recruitment of type I (Allemeier et al., 1994; Jacobs et al., 1987;

Jansson et al., 1990; Pette., 1997; Ross & Leveritt., 2001). Neural impulse responses are responsible for these alterations, due to the progression/ loading of HIT, which alters metabolic homeostasis (Pette., 1985). Further neural adaptations, following 4 weeks of HIT (4-10 x 30sec sprints with 4min recovery, twice a week), are an increased motor unit activation in the vastus lateralis (Creer et al., 2004). Which is in keeping with producing greater amounts of peak power output (PPO), mean power output (MPO) and total work (Creer et al., 2004). It is also thought that following 5 weeks of HIT (20 x 10sec sprint with 50sec recovery, 3 times a week, against 8-8.5% body mass) leads to an increase in sarcoplasmic reticulum, due to an increase in $Ca^{2+}$ release of ~5.5% (Ørtenblad et al., 2000). Who also found greater maintenance of MPO during 10 sprints in pre to post and compared to the control group (Ørtenblad et al., 2000).

### 4.1.2 Maintenance of power

HIT involves a series of repeated sprints of between 6 seconds and 4 minutes duration and rest durations that are longer than the sprint duration. Previous literature has demonstrated that improvements in endurance capacity, endurance performance and power output is tailored around the specific work to rest ratio during HIT (Kavaliauskas, Aspe, Babraj., 2016). For example, Kavaliauskas, Aspe, Babraj., (2016) speculate that their HIT group with the shortest work to rest ratio (1:3) increased in $VO_2$ peak, TTE and TT due to a larger aerobic demand from a shorter rest. However, research is starting to indicate that maintaining power output during HIT leads to similar training adaptations to HIT protocols that use shorter rest and work or reduced work to rest ratios (Hazell et al., 2010; Study 2; Yamagashi & Babraj 2017). Typically, an increase in PCr degradation and fall in peak power during HIT (4-6 x 30sec sprint 4 min recovery) is a key factor for improving endurance, as this creates a greater aerobic metabolism demand during training (Bogdanis et al., 1995; Bogdanis et al., 1996; Gaitanos et al., 1993; Sloth et al., 2013). However, the majority of PCr degradation and glycogen uptake during HIT occurs within the first 15 sec of a 30 sec sprint (Bogdanis et al., 1985; Bogdanis et al., 1998; Parolin et al., 1999). Therefore, similar training adaptations that occur using a 30sec sprint can also occur when reducing the sprint duration by half

(Yamagashi & Babraj., 2017). Maintaining to ~ 60% of maximal power output during HIT (4 x 30sec sprint 4min recovery) causes a continued decrease in PCr and glycogen (McCartney et al., 1986), and as long as there are multiple bouts there will be an increasing demand on oxidative phosphorylation (Hazell et al., 2010; McCartney et al., 1986; Spriet et al., 1989). Potentially explaining the similar endurance adaptations between using longer vs. shorter work to rest ratios and shorter sprints vs. longer sprints (1:8, 1:12, 1:24, 4-6 x 30 and 10sec sprints Hazell et al., 2010; 1:8, 15 and 30sec sprints Yamagashi & Babraj., 2017). With Hazell et al., (2010) finding a decrease in 5km TT (1:8: ~ 5.2%, 1:12: ~ 3%, 1:24: ~ 3.5%) and increase in $VO_2$ max (1:8: ~ 9.3%, 1:12: ~ 3.8%, 1:24: ~ 9.2%), and Yamagashi & Babraj., (2017) finding an increase in $VO_2$ peak (15sec: ~ 12.1%, 30sec: ~ 12.8%), TTE (15sec: ~ 16.2%, 30sec: ~ 12.8%), CP (15sec: ~ 7.8%, 30sec: ~ 7.4%) and decrease in 10km TT (15sec: ~ 8.6%, 30sec: ~ 7.2%).

### 4.1.3 Variables for adaptations

Hazell et al., (2010) used 4-6 cycle sprints (7.5% body mass resistance), with either a 30sec (4 min rest) or 10sec (4 min and 2min rest) sprint duration. Manipulating the work:rest ratio (1:12, 1:24) to maintain peak power output (PPO (1:12 = 95%. 1:24 = 96%)), MPO (1:12 = 82%. 1:24 = 84%) and minimum power output (1:12 = 69%. 1:24 = 73%) could explain the similar pre vs. post testing results in $VO_2$ max (1:8 = 9.3%. 1:12 = 3.8%. 1:24 = 9.2%) and 5km TT run (1:8 = 5.2%. 1:12 = 3%. 1:24 = 3.5%) when using a work:rest ratio (1:8) that saw a diminished PPO (89%), MPO (58%) and minimum power output (40% (Hazell et al., 2010)). The 1:12 group no significant change in $VO_2$ max ($p = 0.06$) despite similar power output data to the 1:24 group. The 1:8 group also saw a greater significant change and percentage change in the 5km TT run test compared to the other training groups (1:8 $p < 0.001$, 1:12 and 1:24 $p < 0.03$). Suggesting that seeking to diminish power output during HIT and create a larger amount of work may have a slight advantage over maintaining power output during HIT. However, similar to Hazell et al., (2010) Yamagashi & Babraj., (2017) compared sprint duration, 15sec (2min rest) and 30sec (4min rest), using 4 cycle sprints before progressing to 6 sprints (7.5% and 6.5% body mass

resistance for males and females respectively) over 18 HIT sessions. Yamagishi & Babraj., (2017) found that using a 15sec and 30sec sprint but with the same work:rest ratio (1:8) found similar training session average PPO percentage change from session 1 (15sec: 1 vs. 6 = 7%, 1 vs. 12 = 7.4%, 1 vs. 18 = 7.9%. 30sec: 1 vs. 6 = 4.6%, 1 vs. 12 = 5.4%, 1 vs. 18 = 5.4%). In addition, the 30sec group had a significant larger average amount of total work (KJ) across all the measured sessions (15sec: < 40 KJ. 30sec: > 55 KJ), with session 12 total work been significantly greater in the 30sec group (~ 57.8 KJ) vs. 15sec group (~ 38.6 KJ). Both training groups experienced similar improvements in $VO_2$ peak (15sec: ~ 12.1%, 30sec: ~ 12.8%), TTE (15sec: ~ 16.2%, 30sec: ~ 12.8%), 10km cycle TT (15sec: ~ 8.6%, 30sec: ~ 7.2%) and CP (15sec: ~ 7.8%, 30sec: ~ 7.4%). Lloyd Jones, Morris, Jakeman., (2017) compared 6sec sprints vs. 30sec sprints both using a work:rest ratio of 1:8 but matched for overall sprint time of 2min (20 x 6sec sprints 48sec recovery, 4 x 30sec sprints 4min recovery, both against 7.5% body mass) for 6 sessions. Both groups produced similar amounts of average PPO across the 6 sessions, with both groups significantly improving PPO in session 6 vs. 1 (6sec = 9%, 30sec = 20%). Across the 6 HIT sessions the 6 sec group produced more total work (~ > 110KJ) compared to the 30sec group (~ < 90KJ), with session 6 total work been significantly greater. In addition to this both groups improved their 10km TT cycle time by a similar amount (6sec = 5.1%, 30sec = 6.2%). Indicating that the reproducibility of power output and not overall total work completed is an important factor for improving endurance using HIT (Lloyd Jones, Morris, Jakeman., 2017). This may explain why Hazell et al., (2010) saw an increase in endurance in all three groups despite the 20sec difference in sprint duration between 1:8 vs. 1:12 and 1:24 work:rest ratio. As the greater work:rest ratio allows a greater maintenance of power output during HIT (Hazell et al., 2010), due a greater recovery of PCr from a longer rest duration (Bogdanis et al., 1985; Bogdanis et al., 1998; Parolin et al., 1999). The intensity of the rest (passive vs. set recovery intensity) during HIT may also play a role on the recovery of PCr (Yamagishi & Babraj., 2016). Yamagishi & Babraj., (2016) found a greater decline in PPO in sprint 2 of 4 x 30sec sprints (4min recovery) when recovering at 30% (~12.7%) and 40% (~12.7%) vs. passive (~7.4%) and 20% (~5.8%) of $VO_2$ peak. Therefore, greater work:rest ratios and

lower intensity of recovery leads to a greater maintenance of power during HIT, perhaps due to a greater recovery in PCr (Bogdanis et al., 1985; Bogdanis et al., 1998; Parolin et al., 1999; Yamagishi & Babraj., 2016).

An explanation for these similar improvements in endurance performance in Yamagishi & Babraj., (2017) could be that the majority of PCr and glycogen uptake occurs within the first 15sec of a 30sec sprint (Bogdanis et al., 1985; Bogdanis et al., 1998; Parolin et al., 1999). Despite the greatest aerobic contribution been present in the final 15sec of a 30sec sprint (Parolin et al., 1999). Further potentially indicating that the aerobic contribution during the sprint may not an important factor for improving endurance after HIT. It is thought that the aerobic demand during the rest periods between sprints may be crucial for improving endurance post HIT (Kavaliauskas, Aspe, Babraj., 2016). Kavaliauskas, Aspe, Babraj., (2016) speculate that creating a shorter work:rest ratio and therefore creating a greater aerobic demand during rest periods could be a factor for causing an increase in endurance adaptations. Suggesting that the duration of the sprint and rest duration is not as great a factor as the work:rest ratio, with a work:rest ratio of 1:8 been consistent in improving endurance adaptations post HIT (Burgomaster et al., 2005, 2006, 2008; Hazell et al., 2010; Kavaliasuskas, Steer, Babraj., 2016; Lloyd Jones, Morris, Jakeman., 2017; Yamagashi & Babraj., 2017).

Manipulating the rest duration between sprints might be another factor to allow the maintenance of MPO (Phillips Thompson, Oliver., 2014; Study 1; Study 2). Phillips., Thompson, Oliver., (2014) speculates that improvements in endurance performance may not be achieved when using self-regulated (SR) rest between 10 x 6 sec cycle sprints (7.5% body mass resistance) due to an over-estimation in rest by 10% which may not stimulate the required aerobic response. However, Study 2 demonstrates a greater percentage change in TTE and 10km TT testing for the SR group vs. fixed rest (30 sec, 1:5 work:rest ratio) group. In addition, although the SR group improved in these endurance tests, there was no increase in $VO_2$ peak, whereas the 30 sec group saw a positive (~ 5%) percentage increase in $VO_2$ peak. This would suggest that the short work to rest ratios in the 30sec group meant that they experienced a larger aerobic demand

than the SR group (Gosselin et al., 2012). The improvement in TTE (~ 3%) and TT (~ 3%) for the SR group could be due to an improvement in anaerobic capacity (Bulbulian et al., 1986; Noakes., 1988). The greater percentage change for the SR group vs. the 30 sec group in PPO (~ 1% vs. ~ -4%) and MPO (~ 0.4% vs. ~ -2%) testing could suggest this is possible. In contrast to the findings of Study 2, Kavaliauskas, Aspe, Babraj., (2016) found that manipulating the rest period between sprints would lead to specific adaptations. Such as shorter rest periods would only lead to improvements in endurance performance and longer rest periods would only lead to improvements in power output. Therefore, it is still uncertain if increasing rest duration between sprints to maintain MPO will lead to any performance adaptation.

### 4.1.4 Sex fatiguing differences

There is consistent research that indicates that females are more fatigue resistant when compared against males (Billaut & Bishop., 2012; Laurent et al., 2010; and Smith & Billaut., 2012). This is reflected by females recruiting a larger amount of type I muscle fibres during HIT whereas males have a larger recruitment in type II muscle fibres (Glenmark et al., 1992; Hicks et al., 2001). This difference in fibre use is strongly correlated to females oxidising more fat during exercise than males (Knechtle et al., 2004). With males demonstrating a higher glycolytic enzyme activity and lower oxidative capacity (Roepstorff et al., 2006; Russ et al 2005) which is in keeping with a greater force development (Russ et al 2005). However, with this greater force development comes a greater disturbance in $Ca^{2+}$ kinetics possibly from an increase in Pi (Glaister., 2005; Gaitanos et al., 1993; Ørtenblad et al., 2011; Westerblad, Allen, Lannergren., 2002). In relation to repeat sprint cycle activity (20 x 5sec sprints 25sec rest against 0.9 N $kg^{-1}$ of body mass), Billaut & Bishop., (2012) found that males had a significant greater power output than females but also saw a greater decrement in power output compared to female participants. They also identify no significant difference between sexes in power output in the final 4 sprints, and identify that females were able to maintain closer to the maximal power output. Indicating that during HIT males experience a greater drop in their performance compared to females. Suggesting that males experience an

impairment in muscular contraction due to diminished $Ca^{2+}$ kinetics (Ørtenblad et al., 2011; Westerblad, Allen, Lannergren., 2002).

### 4.1.5 Possible sex differences in performance adaptation

Due to the female ability to recruit more type I muscle fibres, oxidise more fat and reduce an accumulation in Pi, it would suggest that females would require a shorter self-regulated (SR) rest period. However, Study 1 found no significant difference in SR rest duration between sexes, with males having a shorter mean SR rest period than females. Study 1 also identified that both male and female participants over-estimate their recovery duration by 10% but not 15%. Normalised resting $VO_2$ data from Study 1 shows that male rest 1 and rest 9 data is significantly greater than female data. Curve data from study 1 is similar in rest 1 and 9 in females across all trials. The curve of $VO_2$ in rest 9 in males is higher than rest 1 when SR rest is reduced by 15%. Indicates that females do not experience the same aerobic demand compared to males during SR HIT. It has also been speculated that creating a greater aerobic demand during HIT will lead to an increase in endurance measures (Gaitanos et al 1993; Kavaliauskas, Aspe, Babraj., 2016; Sloth et al., 2013). Therefore, it is unsure if SR HIT, even with a reduction of ≥ 15% will stimulate a response increase in $VO_2$ peak for females. Given that Study 2 found no percentage change in $VO_2$ peak following 6 sessions of SR HIT when using male participants, females may require a larger reduction in SR rest to stimulate a larger aerobic response (Gaitanos et al., 1993).

### 4.1.6 Self-regulated rest

It has been shown that young adult participants (18-35 years) can SR their recovery time effectively to maintain sprint speed performance (12 x 30m (Glaister et al., 2010)) and MPO (10 x 6 seconds, 7.5% body mass resistance (Phillips Thompson, Oliver., 2014; Study 1; Study 2)) to maintain maximal performance (coefficient of variation (CV) ≤ 5.2%)). Both Glaister et al., (2010) and Phillips Thompson, Oliver., (2014) have shown that young adults can self-regulate recovery between sprints. However, in studies 1 and 2 we found that

not all participants managed to do so with a combined failing of 18% in both studies. Glaister et al., (2010) have suggested that participants with a lower level of aerobic capacity would choose longer rest periods, suggesting the longer the rest period would indicate an increase in fatigue, and this could be used as a surrogate indicator for fatigue. However, Phillips Thompson, Oliver., (2014) suggests that participants could be using pacing strategies during SR rest to prevent any homeostatic disturbances that could lead to early exercise termination (Tucker et al., 2006). It is believed that participants pace their efforts to prioritise energy expenditure (Edwards & Polman., 2012). Pre planning will identify what the task is, the importance of the task, the person's capabilities and willingness to do the task (Edwards & Polman 2013).

During self-paced cycling exercise, there is evidence to suggest that the exercise is regulated through sensory feedback to the central nervous system (CNS) through central fatigue (Davis., 1995; Froyd et al 2016; Meeusen et al., 2006; Swart et al 2012; Kay et al 2001; Noakes et al 2001; St Clair Gibson et al 2001; Swart et al 2009; Tucker et al 2006). Central fatigue is defined as a reduction in maximal capacity of the CNS to optimally recruit motor units to produce force (Gandevia., 2001). This is to ensure that within peripheral fatigue the participant's peripheral critical threshold is never exceeded (Amann., 2011; Amann., 2012). The peripheral critical threshold is defined as the reduction in the muscle capacity to beyond the neuromuscular junction to produce maximal force (Froyd et al 2016). It is also thought that peripheral fatigue is a regulator for self-paced cycling exercise by reducing the amount of muscle recruitment through afferent feedback (from peripheral organs: lungs, heart and skeletal muscle (Amann., 2011; Amann., 2012; Froyd et al 2016)). Afferent feedback comes from sensory nerves located within muscle spindles and Golgi tendon organs (Marcora., 2008; Proske., 2005). These sensory nerves sense tension, position and movement, and then send signals through the CNS to give the sense of effort (Proske., 2005). Both central and peripheral fatigue are believed to contribute to neuromuscular fatigue (Froyd et al 2016). It has been demonstrated that when intensity during exercise bouts increase there is also an increase in neuromuscular and peripheral fatigue (Amann & Dempsey., 2008). However, these studies have used self-paced time trial cycling and not

looked at central or peripheral fatigue during rest periods. What can be highlighted from these studies is that peripheral fatigue occurs 20% into the time trial and steadily increases as the time trial continues (Froyd, Millet, Noakes., 2013). Whereas central fatigue is thought to occur when after peripheral fatigue had already developed (Decorte et al., 2012) and only further develops depending on the exercise duration (Place et al., 2010). Giving the lack of research in central and peripheral fatigue during HIT specifically in recovery periods, it would suggest from the above that peripheral fatigue plays a larger role during SR rest. Therefore, SR rest could be regulated by afferent feedback.

Phillips Thompson, Oliver., (2014) and study 1 identified that male and female participants over-estimated their SR rest period by at least 10%. There is a potential that pacing tactics may have occurred in both sexes when SR rest is reduced by 15%, due to the significant drop in MPO when compared to the criterion sprint (~ -5.1%) and trial 3 (~ -4.1% (Study 1)). This could potentially explain how the participants were able to keep their CV% to ≤ 5.2%. There is a possibility that females are using pacing tactics in Study 1 as the criterion MPO data is significantly greater than trials 1-6 (T1: ~ -6.5%. T2: ~ -8.6%. T3: ~ -8.4%. T4: ~ -6.5%. T5: ~ -4.3%. T6: ~ -7.5%). Pacing tactics may have occurred to prevent homeostatic disturbance that would have led to early exercise failure (Tucker et al., 2006). As an observation from studies 1 and 2, participants with an above within group average $VO_2$ peak (Study 1: males = 43 ± 5, females = 33 ± 6 $ml.kg^{-1}.min^{-1}$. Study 2: 48 ± 7 $ml.kg^{-1}.min^{-1}$) and longer TTE (Study 1: males = 718 ± 80, females 511 ± 45secs. Study 2: 544 ± 75secs) times and did not consistently have a shorter rest period than participants who had a below group average $VO_2$ peak (Study 1 sex combined: high $VO_2$ peak = 95 ± 32 secs, low $VO_2$ peak = 106 ± 31 secs. Study 2: high $VO_2$ peak = 103 ± 44 secs, low $VO_2$ peak = 106 ± 43 secs) and TTE (Study 1 sex combined: high TTE = 93 ± 32, low TTE = 108 ± 30secs. Study 2: high TTE = 111 ± 38, low TTE = 98 ± 47secs). This suggests that SR rest duration is a personal choice and argues against Glaister et al., (2010) hypothesis that participants with a greater endurance capacity will select shorter rest periods. Using SR rest could have the potential to create specific rest times for each individual during HIT.

### 4.1.7 Aims and hypothesis

Given that male and female participants over-estimate their required SR rest by 10% (Phillips Thompson, Oliver., 2014; Study 1), this study aimed to determine the impact of removing over recovery on endurance adaptations to 4 weeks of HIT. The second aim was to identify if any post HIT endurance adaptation should be present when reducing each participant's most reliable SR rest time by 15% or 20% between 10 x 6 sec cycle sprints. It was hypothesised that both groups and sexes would see positive performance adaptations in TTE, TT and CP testing by either maintaining close to maximal performance or by experiencing a larger aerobic demand due to the reduction in SR rest. It was also hypothesised that females would not experience a greater increase in $VO_2$ peak compared to males due to their significantly less aerobic activity compared to males when SR rest is reduced by 15% (Study 1).

## 4.2 Methods

### 4.2.1 Participants

Physically active young adult males (n = 24, 180 ± 7 cm, 82 ± 14 kg, and 48 ± 8 $VO_{2peak}$ ml.kg$^{-1}$.min$_{-1}$) and females (n = 24, 166 ± 7 cm, 64 ± 10 kg, and 39 ± 8 $VO_{2peak}$ ml.kg$^{-1}$.min$^{-1}$) volunteered for this study. Participants took part in more than the American College of Sports Medicine and American Heart Association recommended 2.5 hours of moderate physical activity (Haskell., 2007). Participants completed 6 ± 3 h (males = 8 ± 3h, females = 5 ± 3h) of structured physical activity (competitive and recreational), and aged between 18-35. Before taking part participants were given written and verbal instructions about the study prior to giving informed consent. Participants also completed a physical activity readiness questionnaire to ensure there was no known health issues that would put the participants in harm by taking part in this study. Ethical approval was received from Abertay University ethics committee and the study was carried out in line with the declaration of Helsinki.

### 4.2.2 Procedures

#### 4.2.2.1 Sprint warm-up

Participants reported to the laboratory, their body mass (kg) and height (cm) were recoreded using a digital scale (Tanita SA 165A-0950U-3) and digital stadiometer (Seca 264) respectively. In all trials subjects were required to complete a warm up that consisted of 4 min cycling at 60 rpm against 1kg as resistance on a cycle ergometer (Monark peak bike). Once completed a sprint specific warm up was carried out; consisting of 3 x 3 second sprints against 7.5% body mass with an active 45s recovery, cycling at 50-60 rpm (with no resistance), between sprints. Subjects then rested for 4 minutes prior to completing the sprint trials (Phillips Thompson, Oliver., 2014).

#### 4.2.2.2 Trial 1

Participants undertook a MPO test (the criterion sprint) consisting of a single 6 sec cycle sprint against a resistance equal to 7.5% body mass to familiarise them with the procedure and to provide criterion sprint data for comparison with repeated sprint performance (see section 2.2.3.3 for further details on the criterion sprint). Participants then cycled against 1kg for 60 sec at 60rpm before sitting quietly for 5 minutes. After this, the 6 sec sprint was then repeated to identify whether a representative maximal effort was achieved in the first test. If participants achieve a lower MPO in test 2, the result of test 1 will be taken as the participants MPO (Phillips Thompson, Oliver., 2014). If participants achieved a MPO in test 2 that is ≥ 5% greater than test 1, a third test was undertaken (Phillips Thompson, Oliver., 2014). This will be repeated as necessary until MPO no longer increases (Phillips Thompson, Oliver., 2014). In this situation, the best performance by the participant was taken as their MPO.

During each trial participants wore a heart rate monitor (Bioharness 2, Zephyr Technology, MD, USA) and gas mask connected to a gas analyser (Metalyzer 3B gas analyser, Cortex, Leipzig, Germany) throughout the protocol (see section 2.2.5.2 for further details on cardiorespiratory measures). Once participants had completed their criterion sprint (CS) test and rested for 8

minutes, they completed the warm up procedure and prepared for their familiarization trial (trial 1). Prior to beginning exercise, participants were reminded that the aim of the trial is to complete 10 x 6 sec maximal effort sprints (see section 2.2.3.2 for sprint duration rational), with a self-regulated recovery between each sprint that enables the participant to replicate in all ten sprints the performance that they achieved in their CS (see section 2.2.3.4 for self-regulated instructions). Participants then completed 10 x 6 sec cycle sprints against a resistance equal to 7.5% of pre-exercise body mass (see section 2.2.3.1 for body mass resistance rational); participants were blind to any timing apparatus.

#### 4.2.2.3 Trials 2-4

In trials 2-4, the protocol was identical to that completed in trial 1, without the use of CS test. Previous research has indicated that two familiarisation trials are required for reliable self-regulation of repeated sprint exercise to be achieved (Glaister et al 2010). The CS test allows participants to familiarise with the bike and identify how to produce their maximal effort over ≥ 2 sprints. Successful self-regulation was measured by taking the MPO scores across all ten sprints and using a within-trial CV measurement. If CV is ≥ 5.2% it was deemed that participants were unable to self-regulate (Glaister et al 2010; Phillips Thompson, Oliver., 2014; see section 2.2.3.3 for further details on CV measures).

#### 4.2.2.4 Pre testing measures

Testing was performed over 3 days with 48 hours between each test. Consisting of a $VO_2$ peak/ time to exhaustion (TTE), leg and lower back dynamometry, critical power (CP), and 10km time trial (TT) test.

#### 4.2.2.5 Day 1, $VO_2$ peak and time to exhaustion test

$VO_2$ peak and TTE test – Participants were fitted with a mask connected to an online expired gas analyser (Cortex Metamax3B). Participants then mounted

the cycle ergometer (Monark peak bike) and cycled for 4 minutes at 60 W. Immediately following this warm up, power output increased by 30 $W \cdot min^{-1}$ until volitional exhaustion (Jakeman Adamson, Babraj., 2012). Participants were instructed to maintain a cadence of 60 RPM and were verbally encouraged throughout. The test was terminated if a participant dropped below a cadence of 60 RPM for more than 5 sec or if the participant chose to stop. $VO_2$ peak was determined as the highest 30 sec average of $VO_2$ across the test (see section 3.2.2.2 for rational on $VO_2$ peak). The duration of the $VO_2$ peak test was recorded (sec) and was defined as a participant's time to exhaustion (Jakeman, Adamson, Babraj., 2012; Stevens & Dascombe., 2015; see section 3.2.2.2 for TTE testing rational).

#### 4.2.2.6 Day 2, Critical power test

CP test – Participants underwent a warm up consisting of 1 minute cycling at 60 RPM against 1kg. Participants were reminded of what the test involves and had the opportunity to ask any questions. Participants completed a maximal effort for 3 min on a cycle ergometer (Monark peak bike 894) against a fixed resistance of 4.5% of their body mass. Participants were strongly verbally encouraged throughout the test and were blind to any timing apparatus, distance covered, and speed/ RPM in an attempt to prevent pacing. The power output from the last 30 sec of the test was determined as a participant's CP and was measured from the cycle ergometer software (Monark Anaerobic Test Software version 2.24.2, Monark Exercise AB).

CP is a reflection of maximal aerobic capabilities, which estimates the relationship between power output and TTE, and is presented as the highest power output value (Watts) over an extended period of time (Bergstrom et al., 2012). In study 3 a CP test was used to determine maximum aerobic power output (Bergstrom et al., 2012; Jones et al., 2010). The CP test involves a maximum all out effort that is typically 3 min long (Bergstrom et al., 2012; Burnley, Doust, Vanhatalo., 2006; Francis et al., 2010; Vanhatalo, Doust, Burnley., 2008) against a resistance of 4.5% of the participant's body mass (Bergstrom et al., 2012). The purpose of this 3 min duration is to allow depletion

of PCr, inhibited glycolysis, increase an accumulation of fatiguing metabolites (such as Pi), and lead to oxidative phosphorylation for ATP turnover (Jones et al., 2010). To calculate CP, a mean average of the power output from the last 30 sec of the 3 min sprint is used (Bergstrom et al., 2012; Jones et al., 2010). This method for measuring CP has been strongly correlated (r = 0.91-0.97) with the power output from the end of a TTE test, but achieved over a shorter distance of time (Jones et al., 2010). Therefore, the CP is indicated to be a reliable measure for determining maximum aerobic power output (Jones et al., 2010).

#### 4.2.2.7 Day 3, Time trial test

TT test – Participants were fitted with a heart rate monitor and mask connected to an online expired gas analyser. Participants then mounted the cycle ergometer to begin a warm up consisted of 4 min cycling at 60 rpm against 1kg as resistance on a cycle ergometer. Participants were then instructed to complete a self-paced 10km cycle time trial as quickly as possible with a fixed resistance (males – 2kg, females – 1.5kg; see section x for greater description of TT test). Only distance covered was reported to the participants (see section 3.2.2.4 for TT testing rational).

$VO_2$, $VCO_2$ and HR was recorded at each km (Baden et al., 2005; St Clair Gibson, Schabort, Noakes., 2001) using 2 sec averages, due to the uncertainty of when a participant would complete a km, and measures were averaged 6 sec around the time of the completed km (St Clair Gibson, Schabort, Noakes., 2001). These measures were then compared between groups and sexes. Due to the sex differences in $VO_2$ peak, percentage of $VO_2$ peak at each km was also calculated (equation 4.1).

% of $VO_2$ peak = ($VO_2$ km / $VO_2$ peak) * 100

*Equation 4.1: Percentage of $VO_2$ peak at each km. Where $VO_2$ km is the $VO_2$ value at a specific km.*

Percentage of predicted HR max was also calculated for each km between groups and sexes (equation 4.2).

PHR-M = (208 – 0.7) * age (years)
% of PHR-M = (HR km / PHR-M) * 100
*Equation 4.2: Shows formula for predicted HR max and percentage of predicted HR max at each km. Where PHR-M is predicted heart rate max, and HR km is the heart rate value at a specific km.*

This equation for predicting HR max is strongly correlated with actual HR max (r = 0.9) and is not effected by sex or physical activity status (Tanaka, Monahan, Seals., 2001). Percentage change of $VO_2$, $VOC_2$ and HR at each km for pre and post time trial testing (change / pre testing value) *100)).

#### 4.2.2.8 Intervention training and training groups

Participants were stratified into two training groups (15% and 20% reduced rest) based on $VO_2$ peak to ensure similar aerobic capacity between groups, and a control group. Both training groups performed 10 x 6 sec sprints with an altered self-regulated rest period. A mean average rest time was calculated from each participant's most reliable trial (the trial with the lowest CV) from trials 1-4. The two training groups consisted of reducing participant's mean self-regulated rest time between sprints by 15% (Males: n = 8, 181 ± 8 cm, 81 ± 14 kg, and 50 ± 11 $VO_2$ peak $ml.kg^{-1}.min^{-1}$, 98 ± 42 sec rest. Females: n = 8, 171 ± 7 cm, 63 ± 7 kg, and 41 ± 11 $VO_2$ peak $ml.kg^{-1}.min^{-1}$, 73 ± 18 sec rest) and 20% (Males: n = 8, 180 ± 5 cm, 83 ± 11 kg, and 48 ± 7 $VO_2$ peak $ml.kg^{-1}.min^{-1}$, 72 ± 34 sec rest. Females: n = 8, 164 ± 7 cm, 62 ± 6 kg, and 41 ± 7 $VO_{2peak}$ $ml.kg^{-1}.min^{-1}$, 77 ± 29 sec rest). Participants who were unsuccessful in maintaining MPO in at least 2 of the 4 trials within trials 1-4 joined the control group and adhered to their normal training schedule (Males: n = 8, 178 ± 8 cm, 83 ± 17 kg, and 47 ± 5 $VO_2$ peak $ml.kg^{-1}.min^{-1}$. Females: n = 8, 165 ± 6 cm, 69 ± 14 kg, and 34 ± 4 $VO_2$ peak $ml.kg^{-1}.min^{-1}$). Other participants joined the control group when the two

training groups reached 16 participants (8 males and females) within each group.

#### 4.2.2.9 Session 1

The same process that occurred in trial 1 was repeated for session 1. Participants performed another MPO test (criterion sprint number 2). After the 8-minute rest the participants performed the sprint trials warm up before beginning their intervention of 10 x 6 sec cycle sprints with either a reduction in self-regulated mean rest by 15% or 20%. Participants wore a heart rate monitor (bioharness) and gas mask.

#### 4.2.2.10 Session 2-8

Similar to session 1, participants completed the sprint trials warm up procedure and then completed 10 x 6 sec cycle sprints with their own specific rest time, (reduced by15% or 20% depending on group) based off their most reliable mean average self-regulated rest time. In sessions 2-7 participants did not wear a heart rate monitor or wear a gas mask. Participants were only required to wear a heart rate monitor and also wear a gas mask that is connected to a gas analyser in session 8. The purpose of this was to create a comparison between each participant's most reliable trial (from trials 1-4), session 1 and 8, thus identifying any potential training adaptations.

#### 4.2.2.11 Post testing measures

Once all 8 sessions were complete, participants recompleted the same three-day testing process used in the pre testing measures with at least 48h after session 8. Once completed participants were thanked for the contribution and given an explanation of the study based on their results.

### 4.2.3 Fatigue index calculations

MPO was recorded for each sprint throughout the trials and Fatigue index (FI; see section 2.2.3.2 for FI rational) for each trial was calculated using the formula (Fitzsommons et al., 1993):

FI = (100 x [total sprint performance / ideal sprint performance]) – 100.

*Where total sprint performance = sum of MPO from all sprints, and ideal sprint performance = number of sprints x greatest MPO.*

### 4.2.4 Statistical analysis

Statistical analysis for the study was analysed on IBM SPSS Version 22.0 software. Two-way (sex * trial) repeated measures (mixed linear model) analysis of variance (ANOVA) compared the following: mean SR recovery time, MPO, and FI; the CS, subjects' reliable trial (based on within-trial CV for MPO) based on the first four trials and best trial (table 4.1). Two x two way (sex*group*time) ANOVA compared magnitude of change for $VO_{2peak}$, TTE, 10km TT and CP. A further two-way repeated mixed linear (group*sex*time) ANOVA compared mean normalised heart rate, $VO_2$ and $VCO_2$ in best trial, training sessions 1 and 8 for rests 1 vs. 9 and sprints 1 vs. 10. Bonferroni post hoc analysis explored significant main effects. Significant main effects between sexes and groups were further explored by using an independent samples T test. In the case of a significant interaction, the data was split by group and or sex, the ANOVA test was performed again and significant main effects were explored as previously described. If sphericity was violated then Greenhouse-Geisser corrections were used. Magnitude of change between pre and post testing was calculated as post value – pre value / pre value *100. Statistical significance was set up as $p \leq 0.05$, and data are mean with ± standard deviation (SD). Standard error of measurement was employed instead of standard deviation in magnitude of change measurements, due to the larger variability. Using standard error of measurement allowed a true reflection of the magnitude in change error by considering group size. Cohen's D was used to measure effect size between the two training groups. Effect size was defined as trivial (0.0-0.2), small (0.2-0.5), moderate (0.6-1.1), and large (1.2-1.9 (Cohen., 1992)). A Pearson's correlation was used to identify an association between

trial/ session data, magnitude in change testing data, and trial/ session cardiorespiratory response data.

## 4.3 Results

### 4.3.1 Trials 1-4 data

#### 4.3.1.1 Self-regulated recovery time

Table 5.1 shows sex comparison (male n = 24, female n = 24) for SR recovery duration, MPO, FI% and CV% within trials 1-4 and best SR trial from each participant. No significant main effect of SR rest was present between trials ($F_{3.18, 1} = 0.562$, $p > 0.05$), between sexes ($F_{1, 45} = 2.781$, $p > 0.05$) or interaction was present ($F_{3.18, 1} = 0.187$, $p > 0.05$).

#### 4.3.1.2 Self-regulated rest 1 vs. rest 9

A significant main effect was present between trials and rest number ($F_{9, 413.132} = 4.785$, $p < 0.05$), post hoc indicates that trial 3 rest 9 is > than trial 1-3/best SR rest 1 ($p < 0.05$), best SR trial rest 9 is > than trial 2-3/best SR rest 1 ($p < 0.05$). No significant main effect of sex ($F_{1, 48.112} = 2.476$, $p < 0.05$) or significant interaction of trial and rest number*sex ($F_{9, 413.132} = 0.751$, $p < 0.05$) was present.

#### 4.3.1.3 Mean power output

A significant main effect was present for MPO between trials ($F_{2.018, 1} = 4.674$, $p < 0.05$), post hoc indicates that the CS is significantly greater than trials 1-4 ($p < 0.05$). A significant main effect between sex was also present in MPO ($F_{1, 45} = 83.514$, $p < 0.05$), post hoc indicates that male MPO data is significantly greater than female MPO data in the CS and in all trials. No significant interaction was present in MPO ($F_{2.018, 1} = 0.712$, $p > 0.05$).

#### 4.3.1.4 Fatigue index

A significant main effect between trials was present in FI% ($F_{2.178, 1}$, = 6.662, $p < 0.05$), post hoc in indicates that trial 1 FI% is significantly greater than trial 4 and best SR trial ($p < 0.05$). No significant main effect between sexes ($F_{1, 45} = 0.096$, $p > 0.05$) or interaction ($F_{2.178, 1} = 1.566$, $p > 0.05$) was present for FI%.

#### 4.3.1.5 Coefficient variation

A significant main effect was present between trials in CV% ($F_{2.466, 1} = 9.078$, $p < 0.05$), post hoc indicates that trial 1 CV% is significantly greater than trial 3, trial 4 and best SR trial CV% ($p < 0.05$) also trial 2 CV% is significantly greater than best SR trial CV% ($p < 0.05$). A significant main effect was also present between sex ($F_{1, 45} = 4.019$, $p < 0.05$), post hoc cannot indicate where significance lies, however, trial 1 is closest to significance ($p = 0.067$), indicating that male CV% is lower than female CV% in trial 1. No significant interaction was present in CV% ($F_{2.466, 1} = 1.148$, $p > 0.05$).

Table 4.1: Performance variables across the 4 trials of SR rest repeated sprint exercise for both sexes.

| | CS[1] | Trial 1 | | Trial 2 | | Trial 3 | | Trial 4 | | Best SR Trial | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| SR rest time (secs) | | | | | | | | | | | |
| Male | - | 94 ± 43 | | 101 ± 44 | | 100 ± 43 | | 102 ± 44 | | 100 ± 40 | |
| Female | - | 82 ± 40 | | 86 ± 37 | | 94 ± 40 | | 92 ± 42 | | 91 ± 30 | |
| SR rest time (secs) | | Rest 1 | Rest 9 | Rest 1 | Rest 9 | Rest 1 | Rest 9 | Rest 1 | Rest 9 | Rest 1 | Rest 9 |
| Male | - | 83 ± 28 | 99 ± 45 | 81 ± 29 | 95 ± 50 | 76 ± 25 | 98 ± 55[a] | 89 ± 42 | 95 ± 38 | 83 ± 33 | 98 ± 49[b] |
| Female | - | 68 ± 18 | 79 ± 34 | 71 ± 27 | 86 ± 44 | 75 ± 24 | 94 ± 38[a] | 71 ± 27 | 92 ± 29 | 69 ± 20 | 91 ± 32[b] |
| MPO ($W.kg^{-1}$) | | | | | | | | | | | |
| Male | 11.14 ± 1.1*+ | 10.67 ± 1.39* | | 10.79 ± 1.36* | | 10.73 ± 1.2* | | 10.83 ± 1.22* | | 10.93 ± 1.23* | |
| Female | 9.21 ± 1+ | 8.23 ± 1.27 | | 8.33 ± 0.98 | | 8.29 ± 1.08 | | 8.42 ± 0.99 | | 8.53 ± 1.03 | |
| Fatigue index (%) | | | | | | | | | | | |
| Male | - | 15.2 ± 17.3** | | 7.7 ± 8.1 | | 6.3 ± 4.8 | | 5.6 ± 3.3 | | 3.9 ± 1.8 | |
| Female | - | 9.8 ± 7.1** | | 8.4 ± 5.3 | | 6.9 ± 3.8 | | 7.1 ± 4.8 | | 6.6 ± 4.6 | |
| CV % | | | | | | | | | | | |
| Male | - | 4.7 ± 2.6† | | 4.5 ± 2.8‡ | | 3.7 ± 2.1 | | 3.8 ± 1.6 | | 2.6 ± 1.2 | |
| Female | - | 6.8 ± 4.9† | | 5.2 ± 2.6‡ | | 4.3 ± 1.9 | | 4 ± 2.3 | | 3.5 ± 2.3 | |

** Significantly greater than corresponding female data. + Significantly greater than trials 1-4 data. ** Significantly greater than trial 4 and best SR trial. † Significantly greater than trials 3, 4 and best SR trial. ‡ Significantly greater than best SR trial. [a] Significantly greater than trial 1-3/best SR trial rest 1. [b] Significantly greater than 2-3/best SR trial rest 1.*

### 4.3.2 Sessions 1-8 data

#### 4.3.2.1 Best self-regulated recovery and reduced rest duration

Table 4.2 shows sex and group comparison (15% group: male n = 8, female n = 8. 20% group: male n = 8, female n = 8) for rest duration, MPO, FI% and CV% within sessions 1-8 and best SR trial from each participant used within the two training groups. A significant main effect between best SR trial and sessions (time$^{1}$) for rest duration was present ($F_{1, 1} = 202.798$, $p < 0.05$), post hoc indicates that the best SR trial rest duration is significantly greater than the 15% and 20% training groups rest duration ($p < 0.05$). No significant main effect between sexes ($F_{1, 28} = 0.742$, $p > 0.05$) or group ($F_{1, 28} = 0.742$, $p > 0.05$) was present for rest duration. No significant interaction was present for time$^{1}$*sex ($F_{1, 1} = 0.408$, $p > 0.05$), time$^{1}$*group ($F_{1, 1} = 1.044$, $p > 0.05$), sex*group ($F_{1, 28} = 1.784$, $p > 0.05$) and time$^{1}$*sex*group ($F_{1, 1} = 1.472$, $p > 0.05$) for rest duration.

#### 4.3.2.2 Mean power output

No significant main effect between CS, best SR trial and sessions 1-8 (time$^{2}$) for MPO was present ($F_{1.845, 1} = 2.021$, $p > 0.05$), and no significant main effect between groups was present ($F_{1, 28} = 0.042$, $p > 0.05$). However, a significant main effect of MPO between sexes was present ($F_{1, 28} = 46.036$, $p < 0.05$), post hoc indicates that male MPO is significantly greater than female MPO in CS, best SR trial and sessions 1-8 ($p < 0.05$). No significant interaction was present in time$^{2}$*group ($F_{1.845, 1} = 0.391$, $p > 0.05$), time$^{2}$*sex ($F_{1.845, 1} = 0.147$, $p > 0.05$), time$^{2}$*group*sex ($F_{1.845, 1} = 0.349$, $p > 0.05$) and group*sex ($F_{1, 28} = 0.368$, $p > 0.05$).

#### 4.3.2.3 Fatigue index

A significant main effect of FI% between best SR trial and sessions 1-8 (time$^{3}$) was present ($F_{5.065, 1} = 2.475$, $p < 0.05$), post hoc is unable to identify where significance lies. No significant main effect of FI% between groups ($F_{1, 28} = 1.301$, $p > 0.05$) and sexes ($F_{1, 28} = 0.004$, $p > 0.05$) was not present. No significant interaction for group*sex ($F_{1, 28} = 0.906$, $p > 0.05$), time$^{3}$*group ($F_{5.065,}$

$_{1}$ = 1.813, p > 0.05), time$^{3}$*sex ($F_{5.065, 1}$ = 1.099, p > 0.05) and time$^{3}$*group*sex ($F_{5.065, 1}$ = 0.539, p > 0.05) was present.

#### 4.3.2.4 Coefficient variation

A significant main effect of CV% between time$^{3}$ was present ($F_{5.037, 1}$ = 4.857, p < 0.05), post hoc indicates that the best SR trial CV% is significantly less that sessions 1-2 and sessions 4-7 (p < 0.05). No significant main effect of group ($F_{1, 28}$ = 0.892, p > 0.05) or sex ($F_{1, 28}$ = 0.014, p > 0.05) was present in CV%. No significant interaction of group*sex ($F_{1, 28}$ = 0.509, p > 0.05), time$^{3}$*group ($F_{5.037, 1}$ = 0.501, p > 0.05), time$^{3}$*sex ($F_{5.037, 1}$ = 0.558, p > 0.05) and time$^{3}$*group*sex ($F_{5.037, 1}$ = 1.201, p > 0.05).

Table 4.2: Performance variables across the 8 sessions of reduced SR rest and best SR trial repeated sprint exercise for both sexes and groups.

| | **Best SR Trial** | | **CS[2]** | | **Session 1** | | **Session 2** | | **Session 3** | | **Session 4** | | **Session 5** | | **Session 6** | | **Session 7** | | **Session 8** | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Rest (secs) | **15%** | **20%** | **15%** | **20%** | **15%** | **20%** | **15%** | **20%** | **15%** | **20%** | **15%** | **20%** | **15%** | **20%** | **15%** | **20%** | **15%** | **20%** | **15%** | **20%** |
| Male | 113 ± 49ϕ | 86 ± 40ϕ | - | - | 98 ± 42 | 72 ± 33 | 98 ± 42 | 72 ± 33 | 98 ± 42 | 72 ± 33 | 98 ± 42 | 72 ± 33 | 98 ± 42 | 72 ± 33 | 98 ± 42 | 72 ± 33 | 98 ± 42 | 72 ± 33 | 98 ± 42 | 72 ± 33 |
| Female | 84 ± 21ϕ | 92 ± 34ϕ | - | - | 73 ± 18 | 77 ± 29 | 73 ± 18 | 77 ± 29 | 73 ± 18 | 77 ± 29 | 73 ± 18 | 77 ± 29 | 73 ± 18 | 77 ± 29 | 73 ± 18 | 77 ± 29 | 73 ± 18 | 77 ± 29 | 73 ± 18 | 77 ± 29 |
| MPO ($W.kg^{-1}$) | | | | | | | | | | | | | | | | | | | | |
| Male | 11.4 ± 1.46* | 10.73 ± 0.96* | 11.33 ± 1.15* | 11.49 ± 1.19* | 10.96 ± 1.5* | 10.6 ± 0.9* | 10.87 ± 1.43* | 10.74 ± 1.09* | 10.93 ± 1.49* | 10.64 ± 0.73* | 11.11 ± 1.51* | 10.69 ± 1.21* | 11.19 ± 1.43* | 10.78 ± 1* | 10.78 ± 1.83* | 10.86 ± 0.9* | 11.33 ± 1.69* | 10.94 ± 0.81* | 11.26 ± 1.69* | 10.96 ± 0.86* |
| Female | 8.8 ± 0.94 | 8.75 ± 1.29 | 9.18 ± 1.15 | 9.1 ± 1.04 | 8.4 ± 0.79 | 8.39 ± 1.19 | 8.63 ± 0.87 | 8.51 ± 1.48 | 8.33 ± 1.24 | 8.7 ± 1.22 | 8.5 ± 1 | 8.79 ± 1.33 | 8.44 ± 0.67 | 8.87 ± 1.19 | 8.58 ± 0.76 | 9.07 ± 1.08 | 8.77 ± 0.92 | 8.77 ± 1.48 | 8.82 ± 1.02 | 8.85 ± 1.37 |
| Fatigue Index (%) | | | | | | | | | | | | | | | | | | | | |
| Male | 3.1 ± 0.9 | 3.6 ± 1.3 | - | - | 4.8 ± 2.3 | 6.4 ± 4.7 | 6.2 ± 4.5 | 7.5 ± 3.8 | 5.4 ± 3.6 | 7.3 ± 3.7 | 4.3 ± 2.8 | 8 ± 4.6 | 5.3 ± 3.5 | 8.6 ± 5.2 | 8.4 ± 8 | 7.4 ± 4.1 | 5.5 ± 2.8 | 9 ± 4.3 | 4.5 ± 3.5 | 7.8 ± 4.7 |
| Female | 4.7 ± 3.2 | 6.4 ± 5.3 | - | - | 5.4 ± 2.1 | 6.1 ± 3.9 | 7 ± 2.6 | 6.2 ± 3.7 | 7.8 ± 5.9 | 7.8 ± 5.4 | 6 ± 3.4 | 6.4 ± 2.5 | 6.4 ± 3.2 | 6.2 ± 3.5 | 8.3 ± 3.7 | 4.9 ± 3.3 | 6.1 ± 2.2 | 6.2 ± 4.3 | 4.8 ± 1.2 | 7.8 ± 6 |
| CV% | | | | | | | | | | | | | | | | | | | | |
| Male | 2.1 ± 0.4† | 2.3 ± 0.8† | - | - | 3.7 ± 1.8 | 4.2 ± 2.7 | 3.9 ± 1.5 | 4.9 ± 3 | 3.5 ± 2.3 | 4.9 ± 2.4 | 3.7 ± 2 | 5 ± 2.8 | 3.8 ± 2.7 | 5.6 ± 3 | 3.4 ± 2.2 | 4.8 ± 2.1 | 4.5 ± 2.2 | 5.3 ± 2 | 3.7 ± 3.2 | 4.6 ± 2 |
| Female | 2.5 ± 0.6† | 2.8 ± 1.2† | - | - | 3.7 ± 1.2 | 4.8 ± 3 | 5 ± 2.4 | 4.3 ± 2.4 | 5.1 ± 5.2 | 4.9 ± 3 | 3.8 ± 1.8 | 4.4 ± 1.6 | 4.8 ± 2.4 | 4.2 ± 2.6 | 5 ± 2.4 | 3.5 ± 1.9 | 4 ± 1.4 | 4.4 ± 3.2 | 3 ± 0.9 | 5 ± 4.2 |

*ϕ Significantly greater than all sessions rest times in both groups and sexes. * Significantly greater than corresponding female data. † Significantly less than sessions 1-2 and sessions 4-7.*

### 4.3.3 Percentage change measures

#### 4.3.3.1 Critical power

Figure 4.1 shows percentage change measures for all groups (15% group: male n = 8, female n = 8. 20% group: male n = 8, female n = 8. Control group: male n = 8, female n = 8) in critical power, $VO_2$ peak, TTE and 10km TT testing. No significant main effect of pre vs. post testing ($test^1$) for critical power was present ($F_{1, 1} = 2.309$, $p > 0.05$) and no significant main effect was found between sexes ($F_{1, 41} = 0.014$, $p > 0.05$). However, a significant main effect between groups was present ($F_{2, 41} = 3.786$, $p < 0.05$), post hoc indicates that in post testing the 20% group critical power is significantly greater than the control group's ($p < 0.05$). No significant interaction of group*sex ($F_{2, 41} = 0.165$, $p > 0.05$), $test^1$*sex ($F_{1, 1} = 0.014$, $p > 0.05$) and $test^1$*group*sex ($F_{2, 2} = 0.165$, $p > 0.05$) was present in critical power data. However, a significant interaction of $test^1$*group was present ($F_{2, 2} = 3.786$, $p < 0.05$), post hoc indicates that the 20% group post testing data is significantly greater than pre testing data ($p < 0.05$), the 15% group is also approaching significance ($p = 0.062$).

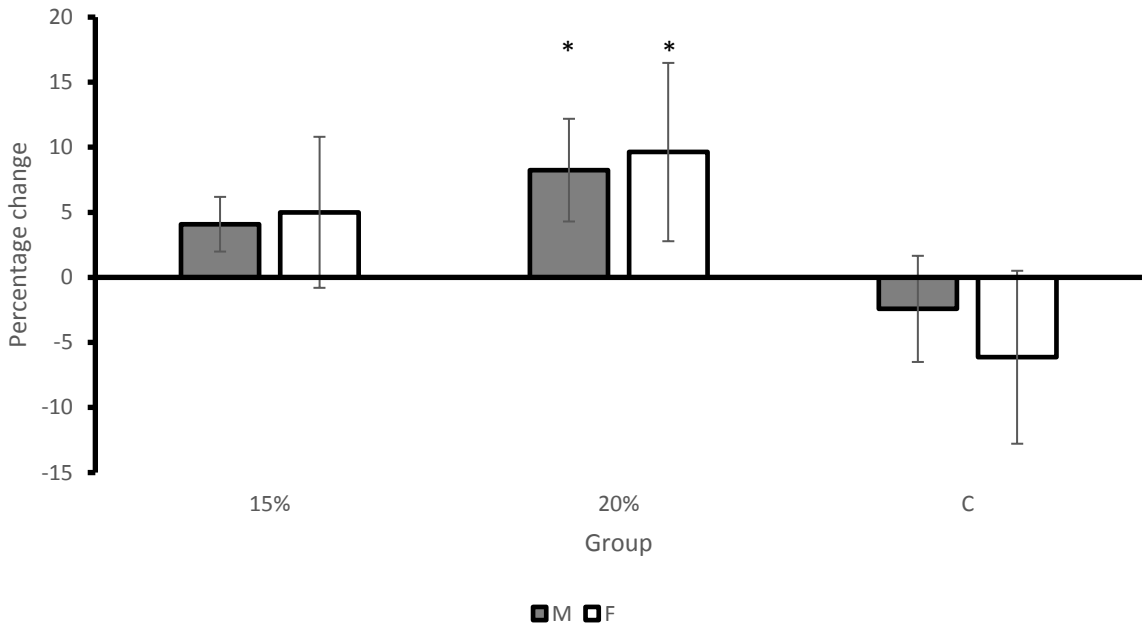


*Figure 4.1: Percentage change ± SEM in CP testing for males and females in the 15% RR, 20% RR and control groups. * Significantly greater than control group data (p < 0.05).*

Table 4.3: Effect sizes between groups for critical power

| Group | M15% | F15% | M20% | F20% | MC | FC |
|---|---|---|---|---|---|---|
| **M15%** | - | d = -0.2 | d = -1.4 | d = -1.2 | d = 2.1 | d = 2.3 |
| **F15%** | d = 0.2 | - | d = -0.7 | d = -0.7 | d = 1.5 | d = 1.8 |
| **M20%** | d = 1.4 | d = 0.7 | - | d = -0.3 | d = 2.7 | d = 2.7 |
| **F20%** | d = 1.2 | d = 0.7 | d = -2.7 | - | d = 2.2 | d = 2.3 |
| **MC** | d = -2.1 | d = -1.5 | d = -2.7 | d = -2.2 | - | d = 0.7 |
| **FC** | d = -2.3 | d = -1.8 | d = -2.7 | d = -2.3 | d = -0.7 | - |

*Table 4.3: M15%, male 15% reduced rest group; F15%, female 15% reduced rest group; M20%, male 20% reduced rest group; F20%, female 20% reduced rest group; MC, male control group; and FC, female control group.*

#### 4.3.3.2 $VO_2$ peak

No significant main effect was present in test[1] for $VO_2$ peak ($F_{1, 1} = 1.73$, $p > 0.05$) and between groups ($F_{2, 42} = 2.743$, $p > 0.05$). However, a significant main effect between sex for $VO_2$ peak was present ($F_{1, 42} = 9.739$, $p < 0.05$); post hoc indicates that within all groups, male $VO_2$ peak is significantly greater than females' in post testing ($p < 0.05$). No significant interaction of group*sex ($F_{2, 42} = 0.181$, $p > 0.05$), test[1]*group ($F_{2, 2} = 2.743$, $p > 0.05$) and test[1]*group*sex ($F_{2, 2} = 0.181$, $p > 0.05$) was present in $VO_2$ peak testing. However, a significant interaction in test[1]*sex was present ($F_{1, 1} = 9.739$, $p < 0.05$), post hoc indicates that males significantly improved $VO_2$ peak greater compared to females in pre to post testing ($p < 0.05$).

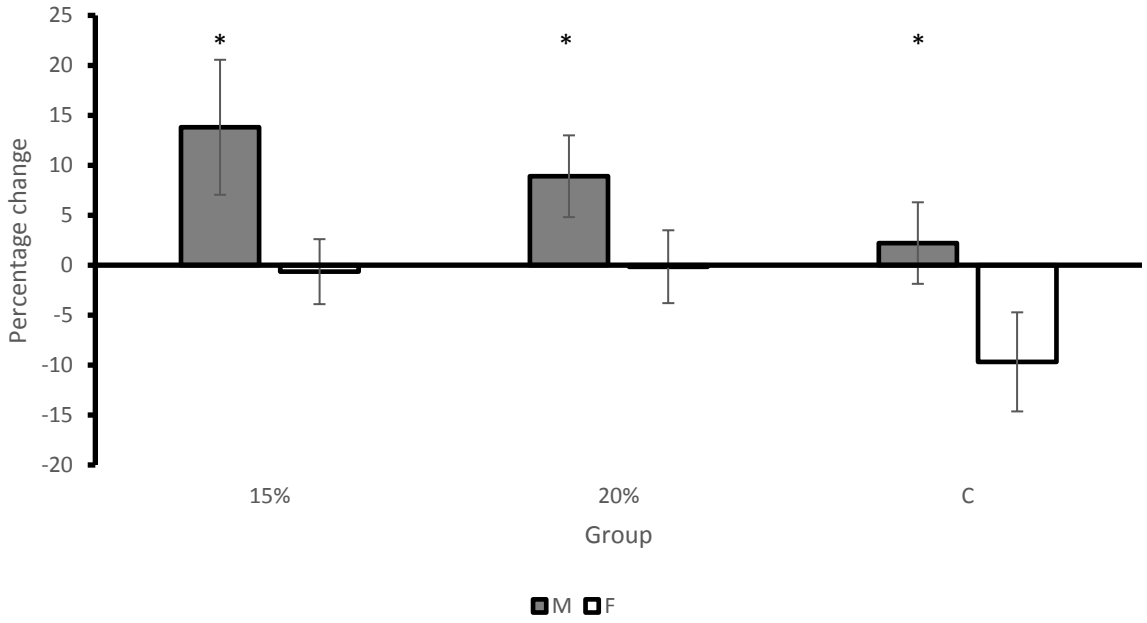


*Figure 4.2: Percentage change ± SEM in $VO_2$ peak testing for males and females in the 15% RR, 20% RR and control groups. * Significantly greater than corresponding female data ($p < 0.05$).*

Table 4.4: Effect sizes between groups for $VO_2$ peak

| Group | M15% | F15% | M20% | F20% | MC | FC |
|---|---|---|---|---|---|---|
| **M15%** | - | d = 2.9 | d = 0.9 | d = 2.7 | d = 2.1 | d = 4 |
| **F15%** | d = -2.9 | - | d = -2.6 | d = -0.1 | d = -0.8 | d = 2.2 |
| **M20%** | d = -0.9 | d = 2.6 | - | d = 2.4 | d = 1.6 | d = 4.1 |
| **F20%** | d = -2.7 | d = 0.1 | d = -2.4 | - | d = -0.6 | d = 2.2 |
| **MC** | d = -2.1 | d = 0.8 | d = -1.6 | d = 0.6 | - | d = 2.6 |
| **FC** | d = -4 | d = -2.2 | d = -4.1 | d = -2.2 | d = -2.6 | - |

#### 4.3.3.3 Time to exhaustion

A significant main effect of test[1] was present in TTE ($F_{1, 1} = 4.063$, $p < 0.05$), post hoc indicates that TTE increased significantly in post testing ($p < 0.05$). No significant main effect between groups ($F_{2, 42} = 0.779$, $p > 0.05$) or sexes ($F_{1, 42} = 0.442$, $p > 0.05$) was present. No significant interaction was present in group*sex ($F_{2, 42} = 0.886$, $p > 0.05$), test[1]*group ($F_{2, 2} = 0.779$, $p > 0.05$), test[1]*sex ($F_{1, 1} = 0.442$, $p > 0.05$) and test[1]*group*sex ($F_{2, 2} = 0.886$, $p > 0.05$).

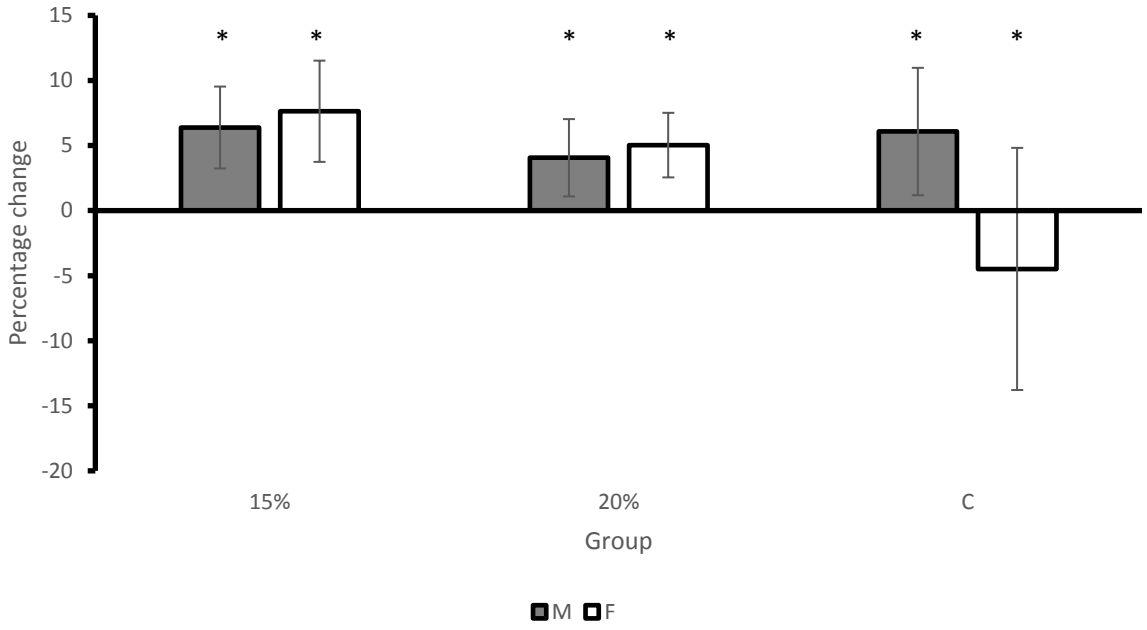


*Figure 4.3: Percentage change ± SEM in TTE testing for males and females in the 15% RR, 20% RR and control groups. * Significant percentage change increase from pre to post testing (p < 0.05).*

Table 4.5: Effect sizes between groups for TTE

| Group | M15% | F15% | M20% | F20% | MC | FC |
|---|---|---|---|---|---|---|
| **M15%** | - | d = -0.3 | d = 0.8 | d = 0.5 | d = 0.1 | d = 1.8 |
| **F15%** | d = 0.3 | - | d = 1 | d = 0.8 | d = 0.3 | d = 1.8 |
| **M20%** | d = -0.8 | d = -1 | - | d = -0.3 | d = -0.5 | d = 1.4 |
| **F20%** | d = -0.5 | d = -0.8 | d = 0.3 | - | d = -0.3 | d = 1.6 |
| **MC** | d = -0.1 | d = -0.3 | d = 0.5 | d = 0.3 | - | d = 1.5 |
| **FC** | d = -1.8 | d = -1.8 | d = -1.4 | d = -1.6 | d = -1.5 | - |

#### 4.3.3.4 Time trial

A significant main effect of test[1] was present in 10km TT ($F_{1, 1} = 24.697$, $p < 0.05$) post hoc indicates that 10km TT decreased significantly in post testing ($p < 0.05$). No significant main effect between groups ($F_{2, 41} = 2.739$, $p > 0.05$) or sexes ($F_{1, 41} = 0$, $p > 0.05$) was present. No significant interaction was present in group*sex ($F_{2, 41} = 0.991$, $p > 0.05$), test[1]*group ($F_{2, 2} = 2.739$, $p > 0.05$), test[1]*sex ($F_{1, 1} = 0$, $p > 0.05$) and test[1]*group*sex ($F_{2, 2} = 0.991$, $p > 0.05$).

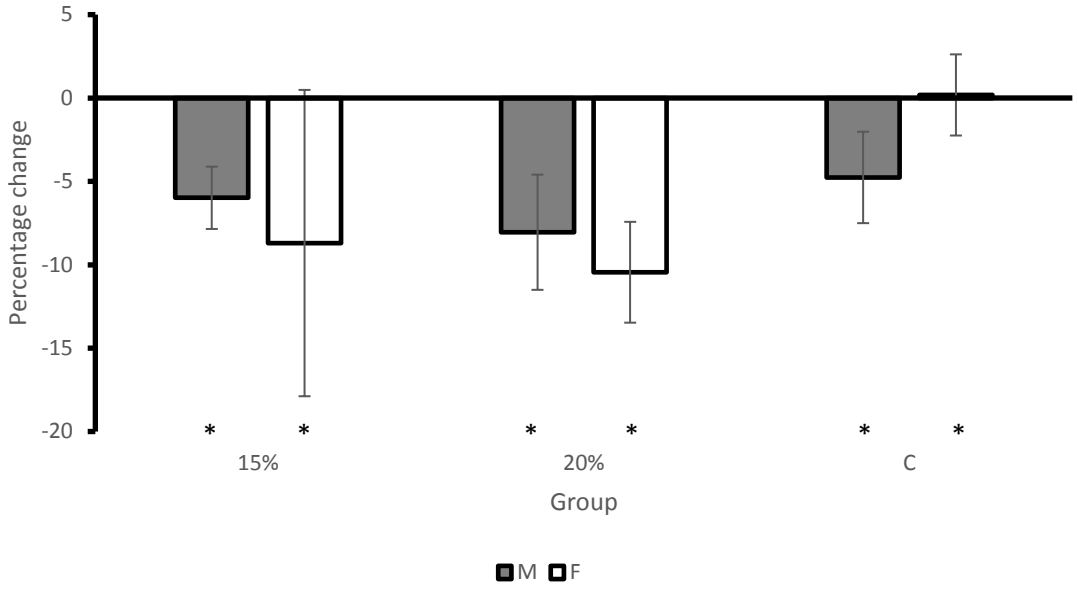


*Figure 4.4: Percentage change ± SEM in TT testing for males and females in the 15% RR, 20% RR and control groups. * Significant percentage change increase from pre to post testing (p < 0.05).*

Table 4.6: Effect sizes between groups for TT

| Group | M15% | F15% | M20% | F20% | MC | FC |
|---|---|---|---|---|---|---|
| **M15%** | - | d = -0.5 | d = -0.7 | d = -1.8 | d = 0.5 | d = 2.9 |
| **F15%** | d = 0.5 | - | d = 0.1 | d = -0.3 | d = 0.7 | d = 1.5 |
| **M20%** | d = 0.7 | d = -0.3 | - | d = -0.7 | d = 1 | d = 2.8 |
| **F20%** | d = 1.8 | d = -0.3 | d = 0.7 | - | d = 2 | d = 3.9 |
| **MC** | d = -0.5 | d = -0.7 | d = -1 | d = -2 | - | d = 2 |
| **FC** | d = -2.9 | d = -1.5 | d = -2.8 | d = -3.9 | d = -2 | - |

#### 4.3.3.5 Correlation

Table 4.7 shows correlation values (r) comparing between the percentage change of in session MPO and the CS (MPO% of CS), in session CV% and FI% for the overall percentage changes of performance tests whilst combining males and females and both training groups' data. No significant correlations occurred in MPO% of CS and: TTE ($r = 0.24$, $p > 0.05$), CP ($r = -0.1$, $p > 0.05$), $VO_2$ peak ($r = 0.21$, $p > 0.05$), and TT ($r = -0.23$, $p > 0.05$). No significant correlations

occurred in CV% and: TTE ($r = -0.03$, $p > 0.05$), CP ($r = 0.3$, $p > 0.05$), $VO_2$ peak ($r = 0.00$, $p > 0.05$), and TT ($r = -0.06$, $p > 0.05$). No significant correlations occurred in FI% and: TTE ($r = -0.02$, $p > 0.05$), CP ($r = 0.3$, $p > 0.05$), $VO_2$ peak ($r = -0.03$, $p > 0.05$), and TT ($r = -0.08$, $p > 0.05$).

Table 4.7: Correlation values comparing between percentage change of in session MPO to the CS and in session CV, and FI.

| Measure | CP | $VO_2$ peak | TTE | TT |
|---|---|---|---|---|
| **MPO% of CS** | r = -0.1 | r = 0.21 | r = 0.24 | r = -0.23 |
| **CV%** | r = 0.3 | r = 0.00 | r = -0.03 | r = -0.06 |
| **FI%** | r = 0.3 | r = -0.03 | r = -0.02 | r = -0.08 |

Table 4.8 shows correlation values for the overall percentage changes of performance tests whilst combining males and females and both training groups' data. Significant correlations lie in: CP vs. TT ($r = -0.47$, $p < 0.05$), TT vs. TTE ($r = -0.43$, $p < 0.05$), and TTE vs. $VO_2$ peak ($r = 0.4$, $p < 0.05$). Tests that did not correlate include: TTE vs. CP ($r = 0.07$, $p > 0.05$), $VO_2$ peak vs. CP ($r = 0.03$, $p > 0.05$), and TT vs. $VO_2$ peak ($r = -0.24$, $p > 0.05$).

Table 4.8: Correlation values between percentage change of performance tests.

| Test | CP | $VO_2$ peak | TTE | TT |
|---|---|---|---|---|
| **CP** | - | r = 0.03 | r = 0.07 | r = -0.47* |
| **$VO_2$ peak** | r = 0.03 | - | r = 0.4* | r = -0.24 |
| **TTE** | r = 0.07 | r = 0.4* | - | r = -0.43** |
| **TT** | r = -0.47* | r = -0.24 | r = -0.43** | - |

***** *Significant positive correlation ($p < 0.05$). ** Significant negative correlation ($p < 0.05$).*

### 4.3.4 Time trial heart rate and gas measures

#### 4.3.4.1 Heart rate

Figure 4.5 shows change of HR in pre and post 10km TT. A significant main effect of HR between km number was present ($F_{19.\ 817.015} = 41.205$, $p < 0.05$). Post hoc indicates that HR is significantly greater in pre 2km vs. pre 1km ($p < 0.05$), pre 3/4km vs. pre 1km and post 1/2km ($p < 0.05$), pre 5km vs. pre 1/2km and post 1/2km ($p < 0.05$), pre 6/7km vs. pre 1/2km and post 1-3km ($p < 0.05$), pre 8km vs. pre 1-4km and post 1-3km ($p < 0.05$), pre 9km vs. pre 1-5km and post 1-4 ($p < 0.05$), pre 10km vs. pre 1-9km and post 1-7km ($p < 0.05$), post 3km vs. pre and post 1km ($p < 0.05$), post 4/5km vs. pre and post 1/2km ($p < 0.05$), post 6km vs. pre 1/2km and post 1-3km, post 7km vs. pre 1-4 and post 1-3km ($p < 0.05$), post 8km vs. pre 1-5km and post 1-4km ($p < 0.05$), post 9km vs. pre 1-6km and post 1-6km ($p < 0.05$), post 10km vs. pre 1-9km and post 1-8km ($p < 0.05$). No significant main effect of group ($F_{2,\ 44.868} = 0.319$, $p > 0.05$) was present. However, a main effect of sex was present ($F_{1,\ 44.868} = 4.231$, $p < 0.05$). Post hoc indicates that female HR data is significantly greater than male HR data in pre 1-3km and post 1/4/7km ($p < 0.05$). No significant interaction of sex*group ($F_{2,\ 44.868} = 1.671$, $p > 0.05$), sex*km number ($F_{19,\ 817.015} = 1.184$, $p > 0.05$) and sex*group*km number ($F_{38,\ 817.013} = 0.976$, $p > 0.05$) was present. However, a significant interaction of group*km number was present ($F_{38,\ 817.013} = 1.715$, $p < 0.05$). Post hoc indicates that the 15% group HR is significantly greater in pre 3/4km vs. pre and post 1km ($p < 0.05$), pre 5/6km vs. pre 1km and post 1/2km ($p < 0.05$), pre 7km vs. pre 1km and post 1-3km ($p < 0.05$), pre 8/9km vs. pre 1-2km and post 1-3km ($p < 0.05$), pre 10km vs. pre 1-4km and post 1-4km ($p < 0.05$), post 4-6km vs. pre 1/2km and post 1/2km ($p < 0.05$), post 7km vs. pre 1km and post 1-3km ($p < 0.05$), post 8/9km vs. pre 1/2km and post 1-3km ($p < 0.05$), post 10km vs. pre 1-6km and post 1-6km ($p < 0.05$); 20% group HR is significantly greater in pre 3/4km vs. pre 1km ($p < 0.05$), pre 5-7km vs. pre 1/2km and post 1km ($p < 0.05$), pre 8/9km vs. pre 1-4km and post 1/2km ($p < 0.05$), pre 10km vs. pre 1-7 and post 1-6km ($p < 0.05$), post 1km vs. pre 1/2km ($p < 0.05$), post 3-5km vs. 1/2km and post 1km ($p < 0.05$), post 6km vs. pre 1/2/4km and post 1km ($p < 0.05$), post 7/8km vs. pre 1-4km and post 1-2km ($p < 0.05$), post 9km vs. pre 1-5km and post 1-3km ($p < 0.05$), post 10km vs.

pre 1-8km and post 1-7km ($p < 0.05$); control group HR is significantly higher in pre 3km vs. post 1km ($p < 0.05$), pre 4-7km vs. post 1/2km ($p < 0.05$), pre 8km vs. pre 1km and post 1/2km ($p < 0.05$), pre 9km vs. pre 1km and post 1-3km ($p < 0.05$), pre 10km vs. pre 1-2km and post 1-4km ($p < 0.05$), post 4km vs. post 1km ($p < 0.05$), post 5/6km vs. post 1/2km ($p < 0.05$), post 7km vs. pre 1km and post 1/2km ($p < 0.05$), post 8 vs. pre 1km and post 1-3km ($p < 0.05$), post 9km vs. pre 1/2km and post 1-3km ($p < 0.05$), post 10km vs. pre 1-5km and post 1-5km ($p < 0.05$).

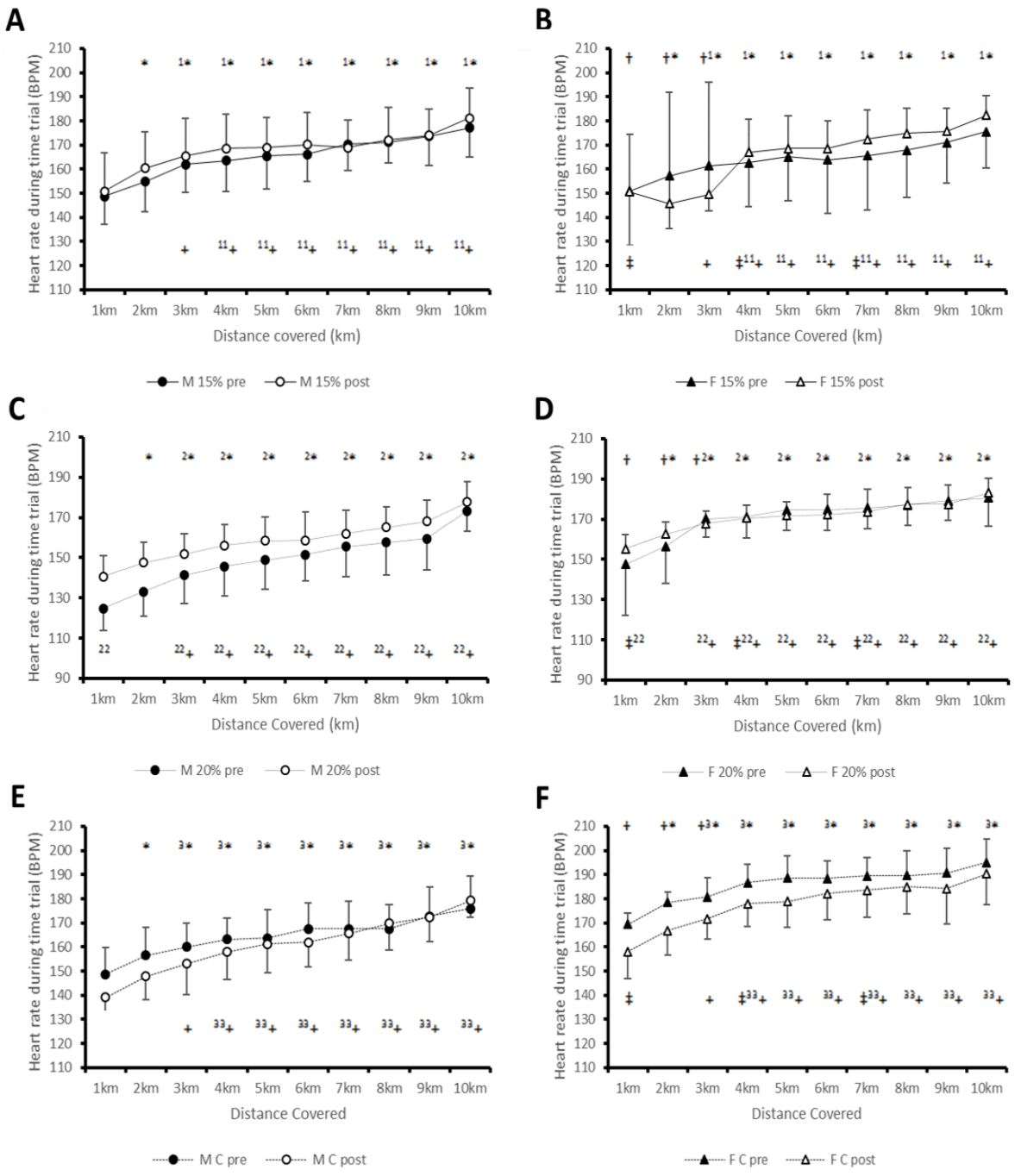

*Figure 4.5: Heart rate during 10km TT in pre vs. post testing at each km for 15% males (A), 15% females (B), 20% males (C), 20% females (D), control males (E) and control females (F). * Significantly greater in pre 2km vs. pre 1km, pre 3/4km vs. pre 1km and post 1/2km, pre 5km vs. pre 1/2km and post 1/2km, pre 6/7km vs. pre 1/2km and post 1-3km, pre 8km vs. pre 1-4km and post 1-3km, pre 9km vs. pre 1-5km and post 1-4, pre 10km vs. pre 1-9km and post 1-7km ($p < 0.05$). + Significantly greater in post 3km vs. pre and post 1km, post 4/5km vs. pre and post 1/2km, post 6km vs. pre 1/2km and post 1-3km, post 7km vs. pre 1-4 and post 1-3km, post 8km vs. pre 1-5km and post 1-4km, post 9km vs. pre 1-6km and post 1-6km, post 10km vs. pre 1-9km and post 1-8km. † Significantly greater than pre male data ($p < 0.05$). ‡ Significantly greater than post male data ($p < 0.05$). [1] Interaction, 15% RR group data is significantly greater in pre 3/4km vs. pre and post 1km, pre 5/6km vs. pre 1km and post 1/2km, pre 7km vs. pre 1km and post 1-3km, pre 8/9km vs. pre 1-2km and post 1-3km, pre 10km vs. pre 1-4km and post 1-4km ($p < 0.05$). [11] Interaction, 15% RR group data is significantly greater in post 4-6km vs. pre 1/2km and post 1/2km, post 7km vs. pre 1km and post 1-3km, post 8/9km vs. pre 1/2km and post 1-3km, post 10km vs. pre 1-6km and post 1-6km ($p < 0.05$). [2] Interaction, 20% RR group data is significantly greater in pre 3/4km vs. pre 1km,pre 5-7km vs. pre 1/2km and post 1km, pre 8/9km vs. pre 1-4km and post 1/2km, pre 10km vs. pre 1-7 and post 1-6km. [22] Interaction, 20% RR group data is significantly greater in post 1km vs. pre 1/2km, post 3-5km vs. 1/2km and post 1km, post 6km vs. pre 1/2/4km and post 1km, post 7/8km vs. pre 1-4km and post 1-2km, post 9km vs. pre 1-5km and post 1-3km, post 10km vs. pre 1-8km and post 1-7km. [3] Interaction, control group data is significantly greater in pre 3km vs. post 1km, pre 4-7km vs. post 1/2km, pre 8km vs. pre 1km and post 1/2km, pre 9km vs. pre 1km and post 1-3km, pre 10km vs. pre 1-2km and post 1-4km($p < 0.05$). [33] Interaction, control group data is significantly greater in post 4km vs. post 1km, post 5/6km vs. post 1/2km, post 7km vs. pre 1km and post 1/2km, post 8 vs. pre 1km and post 1-3km, post 9km vs. pre 1/2km and post 1-3km, post 10km vs. pre 1-5km and post 1-5km ($p < 0.05$).*

Figure 4.6 shows percentage of HR max in pre and post 10km TT. A main effect of km number was present ($F_{19, 817.047} = 40.416$, $p < 0.05$). Post hoc indicates that percentage of HR max is greater in pre 2km vs. pre and post 1 km ($p < 0.05$), pre 3/4km vs. pre 1km and post 1/2km ($p < 0.05$), pre 5/6km vs. pre 1/2km and post 1/2km ($p < 0.05$), pre 7km vs. pre 1/2km and post 1-3km ($p < 0.05$), pre 8km vs. pre 1-4km and post 1-3km ($p < 0.05$), pre 9km vs. pre 1-5km and post 1-4km ($p < 0.05$), pre 10km vs. pre 1-8km and post 1-7km ($p < 0.05$), post 3km vs. pre and post 1km ($p < 0.05$), post 4/5km vs. pre and post 1-2km ($p < 0.05$), post 6km vs. pre 1/2km and post 1-3km ($p < 0.05$), post 7km vs. pre 1-4km and post 1-3km ($p < 0.05$), post 8km vs. pre 1-5km and post 1-4km ($p < 0.05$), post 9km vs. pre 1-6km and post 1-6km ($p < 0.05$), post 10km vs. pre 1-9km and post 1-8km ($p < 0.05$). Also, a significant main effect of sex was present ($F_{1, 44.875} = 4.255$, $p < 0.05$). Post hoc indicates that female percentage of HR max data is significantly greater than male data in pre 1-3km and post 1/4/7km ($p < 0.05$). No significant main effect of group was present ($F_{2, 44.875} = 0.335$, $p > 0.05$). No significant interaction of sex*group ($F_{2, 44.875} = 1.805$, $p > 0.05$), sex*km ($F_{19, 817.047} = 1.169$, $p > 0.05$) and sex*group*km ($F_{38, 817.044} = 0.986$, $p > 0.05$) was present. However, a significant interaction of group*km was present ($F_{38, 817.044} = 1.671$, $p < 0.05$). Post hoc indicates that 15% group percentage of HR max data is significantly greater in pre 3/4km vs. pre and post 1km ($p < 0.05$), pre 5-7km vs. pre 1km and post 1/2km ($p < 0.05$), pre 8/9km vs. pre 1/2km and post 1-3km ($p < 0.05$), pre 10km vs. pre 1-4km and post 1-3km ($p < 0.05$), post 4km vs. pre and post 1km ($p < 0.05$), post 5-7km vs. pre 1km and post 1/2km ($p < 0.05$), post 8/9km vs. pre 1/2km and post 1-3km ($p < 0.05$), post 10km vs. pre 1-6km and post 1-5km ($p < 0.05$); 20% group percentage of HR max data is significantly greater in pre 3/4km vs. pre 1km ($p < 0.05$), pre 5-7km vs. pre 1/2km and post 1km ($p < 0.05$), pre 8km vs. pre 1/2/4km and post 1km ($p < 0.05$), pre 9km vs. pre 1-4km and post 1/2km ($p < 0.05$), pre 10km vs. pre 1-7km and post 1-4km ($p < 0.05$), post 2km vs. pre 1km ($p < 0.05$), post 3-6km vs. pre1/2km and post 1km ($p < 0.05$), post 7km vs. pre 1/2/4km and post 1km ($p < 0.05$), post 8km vs. pre 1-4km and post 1/2km ($p < 0.05$), post 9km vs. pre 1-4km and post 1/2km ($p < 0.05$), post 10km vs. pre 1-8 and post 1-7km ($p < 0.05$); control group percentage of HR max data is significantly greater in pre 3km vs. post 1km ($p < 0.05$), pre 4-7km vs. post 1/2km ($p < 0.05$), pre 8km vs.

pre 1km and post 1/2km ($p < 0.05$), pre 9km vs. pre 1km and post 1-3km ($p < 0.05$), pre 10km vs. pre 1/2km and post 1-4km ($p < 0.05$), post 3/4km vs. post 1km ($p < 0.05$), post 5/6km vs. post 1/2km ($p < 0.05$), post 7km vs. pre 1km and post 1/2km ($p < 0.05$), post 8km vs. pre 1km and post 1-3km ($p < 0.05$), post 9km vs. pre 1/2km and post 1-3km ($p < 0.05$), post 10km vs. pre 1-5km and post 1-5km ($p < 0.05$).

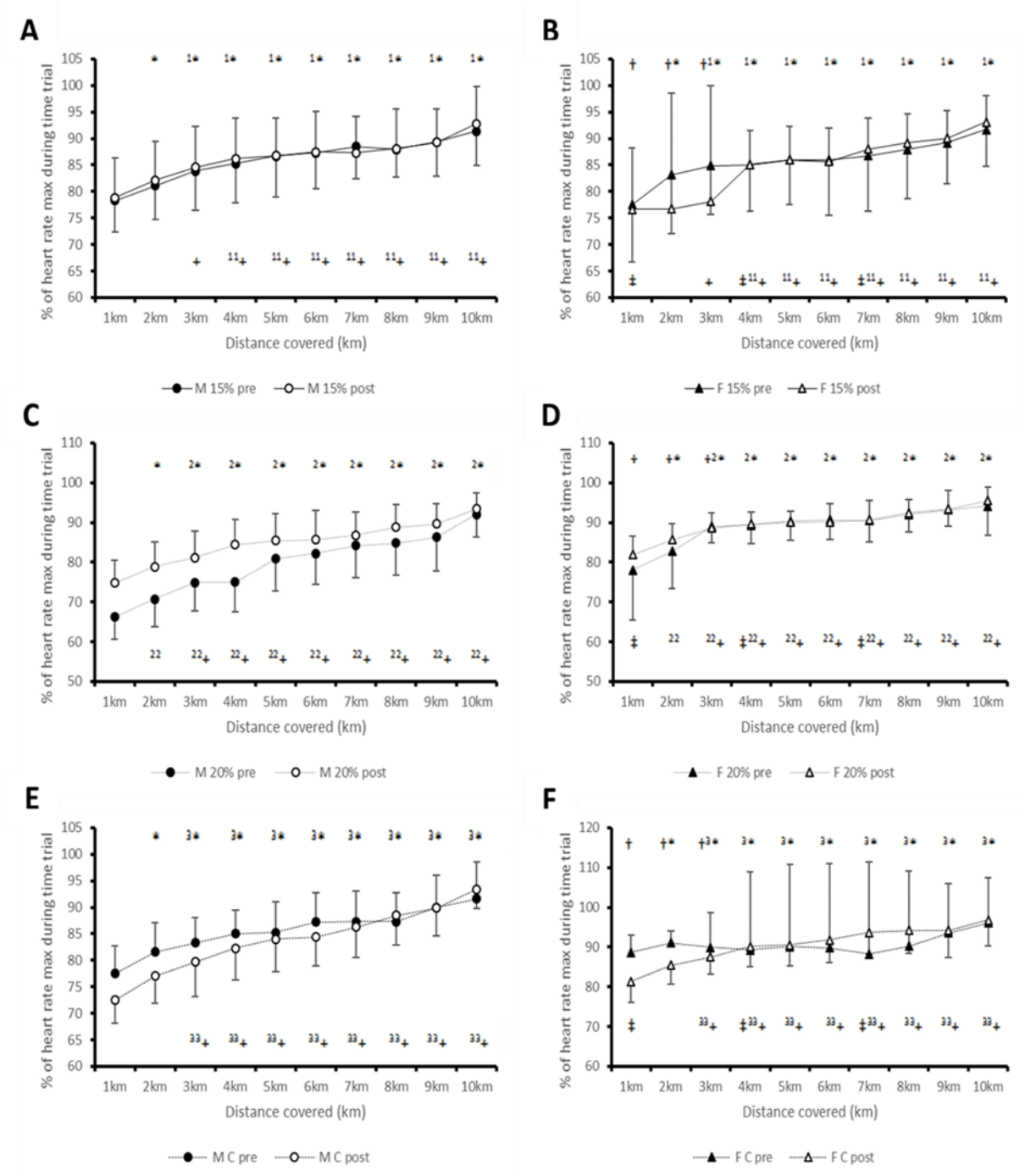


*Figure 4.6: Percentage of heart rate max during 10km TT in pre vs. post testing at each km for 15% males (A), 15% females (B), 20% males (C), 20% females*

*(D), control males (E) and control females (F). * Significantly greater in pre 2km vs. pre and post 1 km, pre 3/4km vs. pre 1km and post 1/2km, pre 5/6km vs. pre 1/2km and post 1/2km, pre 7km vs. pre 1/2km and post 1-3km, pre 8km vs. pre 1-4km and post 1-3km, pre 9km vs. pre 1-5km and post 1-4km, pre 10km vs. pre 1-8km and post 1-7km ($p < 0.05$). + Significantly greater in post 3km vs. pre and post 1km, post 4/5km vs. pre and post 1-2km, post 6km vs. pre 1/2km and post 1-3km, post 7km vs. pre 1-4km and post 1-3km, post 8km vs. pre 1-5km and post 1-4km, post 9km vs. pre 1-6km and post 1-6km, post 10km vs. pre 1-9km and post 1-8km ($p < 0.05$). † Significantly greater than pre male data ($p < 0.05$). ‡ Significantly greater than post male data ($p < 0.05$). [1] Interaction, 15% RR group data is significantly greater in pre 3/4km vs. pre and post 1km, pre 5-7km vs. pre 1km and post 1/2km, pre 8/9km vs. pre 1/2km and post 1-3km, pre 10km vs. pre 1-4km and post 1-3km ($p < 0.05$). [11] Interaction, 15% RR group data is significantly greater in post 4km vs. pre and post 1km, post 5-7km vs. pre 1km and post 1/2km, post 8/9km vs. pre 1/2km and post 1-3km, post 10km vs. pre 1-6km and post 1-5km ($p < 0.05$). [2] Interaction, 15% RR group data is significantly greater in pre 3/4km vs. pre 1km, pre 5-7km vs. pre 1/2km and post 1km, pre 8km vs. pre 1/2/4km and post 1km, pre 9km vs. pre 1-4km and post 1/2km, pre 10km vs. pre 1-7km and post 1-4km ($p < 0.05$). [22] Interaction, 15% RR group data is significantly greater in post 2km vs. pre 1km, post 3-6km vs. pre1/2km and post 1km, post 7km vs. pre 1/2/4km and post 1km, post 8km vs. pre 1-4km and post 1/2km, post 9km vs. pre 1-4km and post 1/2km, post 10km vs. pre 1-8 and post 1-7km ($p < 0.05$). [3] Interaction, control group data is significantly greater in pre 3km vs. post 1km, pre 4-7km vs. post 1/2km, pre 8km vs. pre 1km and post 1/2km, pre 9km vs. pre 1km and post 1-3km, pre 10km vs. pre 1/2km and post 1-4km ($p < 0.05$). [33] Interaction, control group data is significantly greater in post 3/4km vs. post 1km, post 5/6km vs. post 1/2km, post 7km vs. pre 1km and post 1/2km, post 8km vs. pre 1km and post 1-3km, post 9km vs. pre 1/2km and post 1-3km, post 10km vs. pre 1-5km and post 1-5km ($p < 0.05$).*

#### 4.3.4.2 $VO_2$

Figure 4.7 shows change of $VO_2$ in pre and post 10km TT. A significant main effect of $VO_2$ between km number was present ($F_{19, 808.319} = 22.169$, $p < 0.05$). Post hoc indicates that $VO_2$ is significantly greater in pre 9km vs. pre 1/2km ($p < 0.05$), pre 10km vs. 1-8km and post 1/2km ($p < 0.05$), post 3km vs. pre 1/2 ($p < 0.05$), post 4/6km vs. pre 1-6km and post 1km ($p < 0.05$), post 5km vs. pre 1-6km and post 1/2km ($p < 0.05$), post 7/8km vs. pre 1-8km and post 1/2km ($p < 0.05$), post 9 vs. pre 1-9km and post 1-3km ($p < 0.05$), post 10km vs. pre 1-10km and post 1-9km ($p < 0.05$). There was also a significant main effect of sex ($F_{1, 44.083} = 123.653$, $p < 0.05$); post hoc indicates that male $VO_2$ data is significantly greater than female $VO_2$ data during each km in pre and post TT testing ($p < 0.05$). No significant main effect between groups was present ($F_{2, 33} = 1.29$, $p > 0.05$). No significant interaction of sex*group ($F_{2, 33} = 0.403$, $p > 0.05$) and group*sex*km ($F_{6.231, 2} = 1.035$, $p > 0.05$) was present. A significant interaction of sex*km was present ($F_{3.115, 1} = 2.231$, $p < 0.05$). Post hoc indicates that male $VO_2$ data is significantly greater in pre 9km vs. pre 1km ($p < 0.05$), pre 10km vs. pre 1-8km and post 1/2km ($p < 0.05$), post 3km vs. pre 1km ($p < 0.05$), post 4km vs. pre 1/2 and post 1km ($p < 0.05$), post 5km vs. pre 1-4km and post 1km ($p < 0.05$), post 6km vs. 1-3km and post 1km ($p < 0.05$), post 7/8km vs. pre 1-6km and post 1/2km ($p < 0.05$), post 9km vs. pre 1-7km and post 1/2km ($p < 0.05$), post 10km vs. pre 1-10km and post 1-9km ($p < 0.05$); female $VO_2$ data is significantly greater in pre 10km vs. pre 1-2km ($p < 0.05$), post 4-8km vs. pre 1/2km ($p < 0.05$), post 9km vs. pre 1-8km and post 1km ($p < 0.05$), post 10km vs. pre 1-9km and post 1-3/5-8km ($p < 0.05$). Also, a significant interaction of group*km was present ($F_{38, 808.316} = 1.506$, $p < 0.05$). Post hoc indicates that 15% group $VO_2$ is greater than pre 10km and vs. pre 1/2km ($p < 0.05$), post 3 vs. pre 1km ($p < 0.05$), post 4km vs. pre 1/2/6 ($p < 0.05$), post 5km vs. pre 1-8km ($p < 0.05$), post 6/7km vs. pre 1/2km ($p < 0.05$), post 8km vs. pre 1/2/4-6km ($p < 0.05$), post 9km vs. pre 1-8km and post 1km ($p < 0.05$), post 10km vs. pre 1-10km and post 1-3km ($p < 0.05$); 20% group $VO_2$ data is significantly greater in pre 10km vs. pre 1-6km ($p < 0.05$), post 4km vs. pre 1-3km ($p < 0.05$), post 5/6km vs. pre 1km ($p < 0.05$), post 7/8km vs. pre 1-4km ($p < 0.05$), post 9km vs. 1-6km ($p < 0.05$), post 10km vs. pre 1-9km and post 1-9km ($p < 0.05$); control

group $VO_2$ data is significantly greater in pre 9km vs. post 1km ($p < 0.05$), pre 10km vs. pre and post 1-2km ($p < 0.05$), post 7/8km vs. post 1km ($p < 0.05$), post 9km vs. pre and post 1/2km ($p < 0.05$), post 10km vs. pre 1-9km and post 1-6/8km ($p < 0.05$).

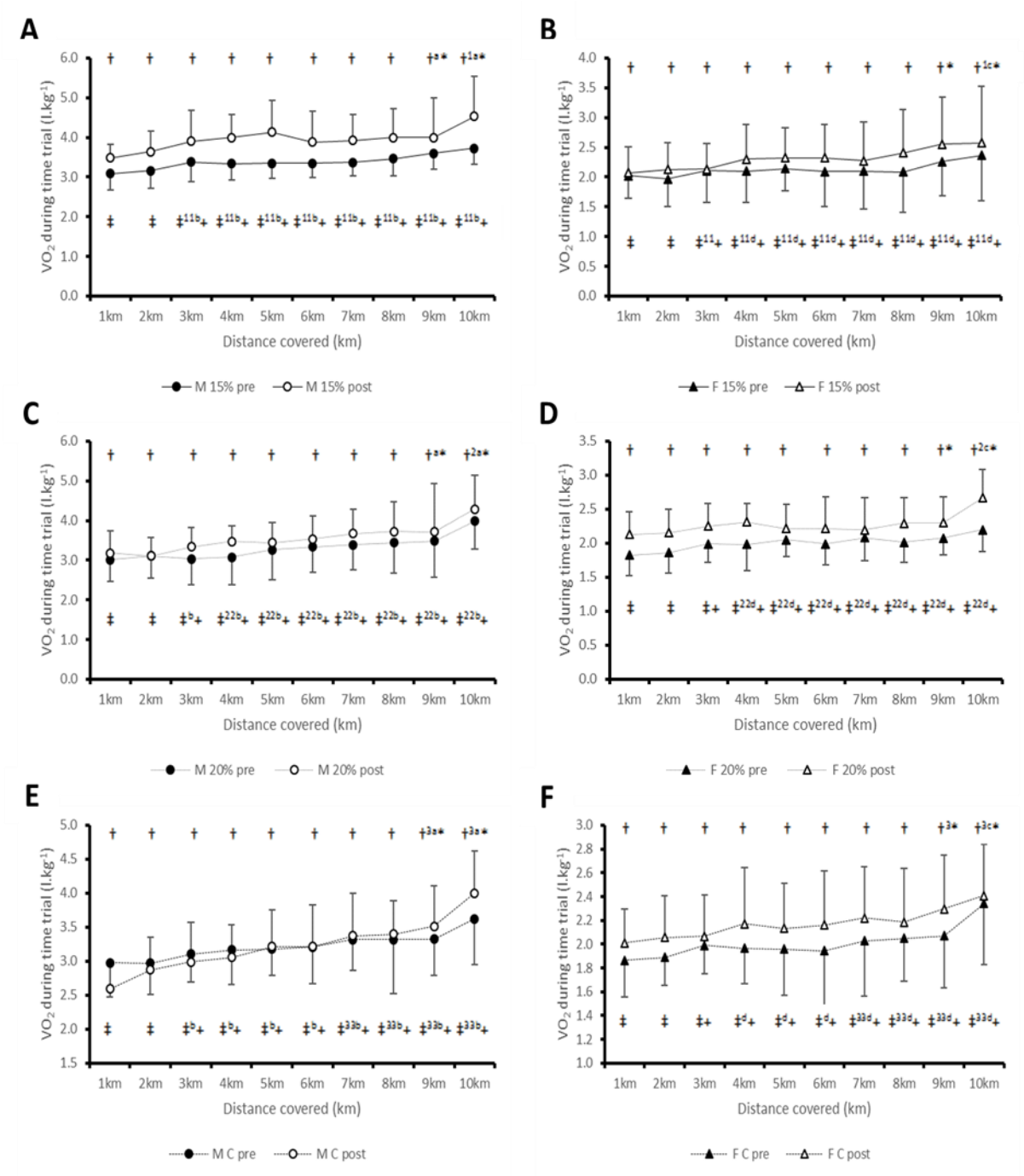


*Figure 4.7: $VO_2$ rate during 10km TT in pre vs. post testing at each km for 15% males (A), 15% females (B), 20% males (C), 20% females (D), control males (E) and control females (F). * Significantly greater in pre 9km vs. pre 1/2km, pre 10km vs. 1-8km and post 1/2km ($p < 0.05$). + Significantly greater in post 3km*

*vs. pre 1/2, post 4/6km vs. pre 1-6km and post 1km, post 5km vs. pre 1-6km and post 1/2km, post 7/8km vs. pre 1-8km and post 1/2km, post 9 vs. pre 1-9km and post 1-3km, post 10km vs. pre 1-10km and post 1-9km ($p < 0.05$). † Significantly greater than pre female data ($p < 0.05$). ‡ Significantly greater than post female data ($p < 0.05$). [a] Interaction, male data is significantly greater in pre 9km vs. pre 1km, pre 10km vs. pre 1-8km and post 1/2km ($p < 0.05$). [b] Interaction, male data is significantly greater in post 3km vs. pre 1km, post 4km vs. pre 1/2 and post 1km, post 5km vs. pre 1-4km and post 1km, post 6km vs. 1-3km and post 1km, post 7/8km vs. pre 1-6km and post 1/2km, post 9km vs. pre 1-7km and post 1/2km, post 10km vs. pre 1-10km and post 1-9km ($p < 0.05$). [c] Interaction, female data is significantly greater in pre 10km vs. pre 1-2km ($p < 0.05$). [d] Interaction, female data is significantly greater in post 4-8km vs. pre 1/2km, post 9km vs. pre 1-8km and post 1km, post 10km vs. pre 1-9km and post 1-3/5-8km ($p < 0.05$). [1] Interaction, 15% RR group data is significantly greater in pre 10km and vs. pre 1/2km ($p < 0.05$). [11] Interaction, 15% RR group data is significantly greater in post 3 vs. pre 1km, post 4km vs. pre 1/2/6, post 5km vs. pre 1-8km, post 6/7km vs. pre 1/2km, post 8km vs. pre 1/2/4-6km, post 9km vs. pre 1-8km and post 1km, post 10km vs. pre 1-10km and post 1-3km ($p < 0.05$). [2] Interaction, 20% RR group data is significantly greater in pre 10km vs. pre 1-6km ($p < 0.05$). [22] Interaction, 20% RR group data is significantly greater in post 4km vs. pre 1-3km, post 5/6km vs. pre 1km, post 7/8km vs. pre 1-4km, post 9km vs. 1-6km, post 10km vs. pre 1-9km and post 1-9km ($p < 0.05$). [3] Interaction, control group data is significantly greater in pre 9km vs. post 1km, pre 10km vs. pre and post 1-2km ($p < 0.05$). [33] Interaction, control group data is significantly greater in post 7/8km vs. post 1km, post 9km vs. pre and post 1/2km, post 10km vs. pre 1-9km and post 1-6/8km ($p < 0.05$).*

Figure 4.8 shows percentage of $VO_2$ peak in pre and post 10km TT. A main effect of km number was present ($F_{19, 808.63} = 12.95$, $p < 0.05$). Post hoc indicates that percentage of $VO_2$ peak is significantly greater in pre 9km vs. pre 1/2km ($p < 0.05$), pre 10km vs. pre 1-8km and post 1-3km ($p < 0.05$), post 4/5/7km vs. pre 1/2km and post 1km ($p < 0.05$), post 6km vs. pre 1/2km ($p <$

0.05), post 8km vs. pre 1-3km and post 1km ($p < 0.05$), post 9km vs. pre 1-8km and post 1/2km ($p < 0.05$), post 10km vs. pre 1-9km and post 1-9km ($p < 0.05$). No significant main effect of sex ($F_{1, 44.25} = 0.002$, $p > 0.05$) and group was present ($F_{2, 44.253} = 0.55$, $p > 0.05$). No significant interaction of group*km ($F_{38, 808.625} = 0.42$, $p > 0.05$), sex*group ($F_{2, 44.253} = 1.017$, $p > 0.05$) and sex*group*km ($F_{38, 808.625} = 0.552$, $p > 0.05$) was present. However, a significant interaction of sex*km was present ($F_{19, 808.63} = 3.988$, $p < 0.05$). Post hoc indicates that male percentage of $VO_2$ peak data is significantly greater in pre 5-7km vs. post 1km ($p < 0.05$), pre 8km vs. post 1/2km ($p < 0.05$), pre 9km vs. pre 1km and post 1/2km ($p < 0.05$), pre 10km vs. pre 1-8km and post 1-8km ($p < 0.05$), post 5/7km vs. post 1km ($p < 0.05$), post 8/9km vs. post 1/2km ($p < 0.05$), post 10km vs. pre 1-9km and post 1-9km ($p < 0.05$); female percentage of $VO_2$ peak data is significantly greater in post 4-8km vs. pre 1/2km ($p < 0.05$), post 9km vs. pre 1-8km ($p < 0.05$), post 10km vs. pre 1-9km and post 1-3km ($p < 0.05$).

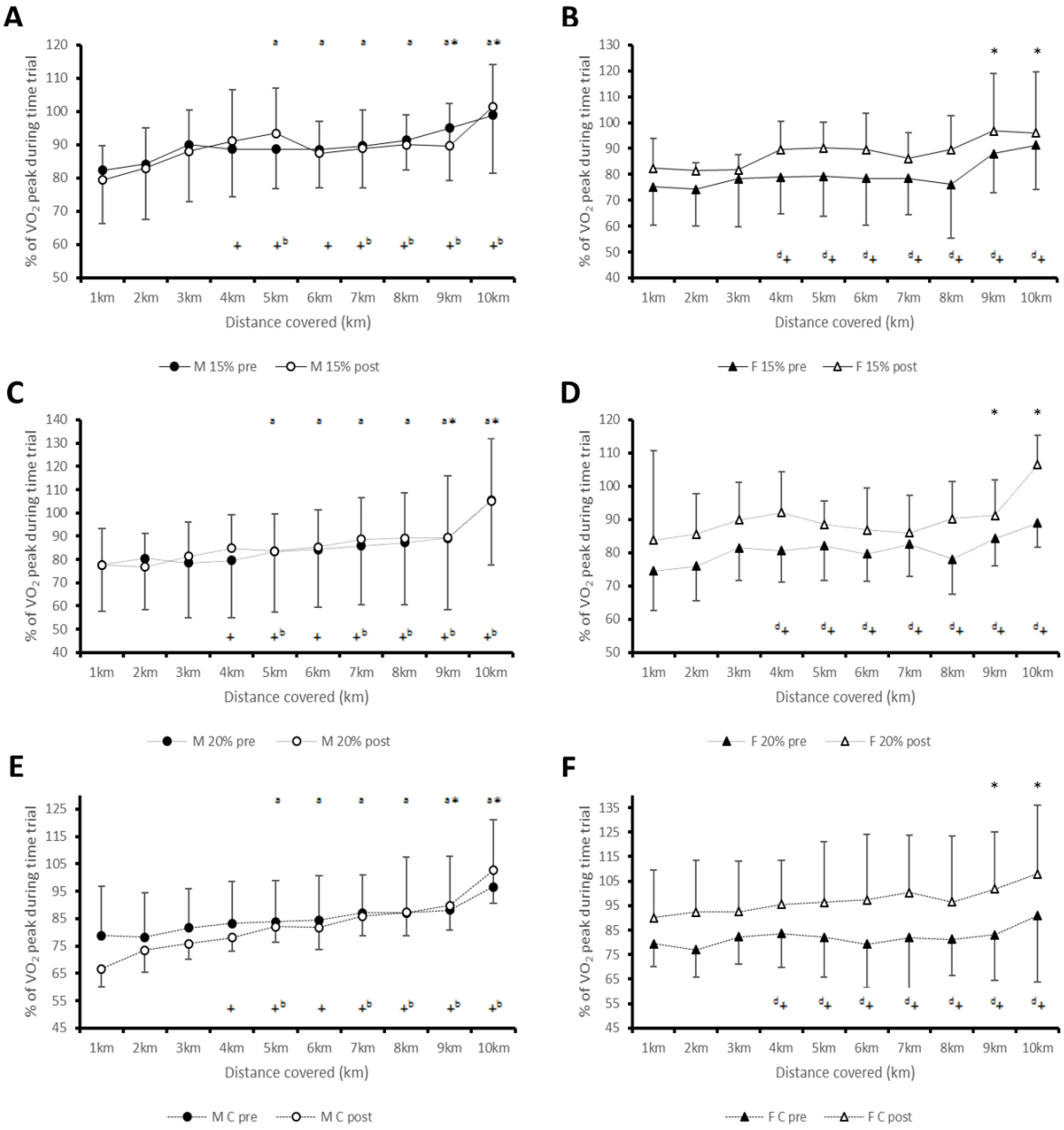


*Figure 4.8: Percentage of $VO_2$ peak during 10km TT in pre vs. post testing at each km for 15% males (A), 15% females (B), 20% males (C), 20% females (D), control males (E) and control females (F). * Significantly greater in pre 9km vs. pre 1/2km, pre 10km vs. pre 1-8km and post 1-3km ($p < 0.05$). + Significantly greater in post 4/5/7km vs. pre 1/2km and post 1km, post 6km vs. pre 1/2km, post 8km vs. pre 1-3km and post 1km, post 9km vs. pre 1-8km and post 1/2km, post 10km vs. pre 1-9km and post 1-9km ($p < 0.05$). [a] Interaction, male data is significantly greater in pre 5-7km vs. post 1km, pre 8km vs. post 1/2km, pre 9km vs. pre 1km and post 1/2km, pre 10km vs. pre 1-8km and post 1-8km ($p < 0.05$). [b] Interaction, male data is significantly greater in post 5/7km*

*vs. post 1km, post 8/9km vs. post 1/2km, post 10km vs. pre 1-9km and post 1-9km ($p < 0.05$). [d] Interaction, female data is significantly greater in post 4-8km vs. pre 1/2km, post 9km vs. pre 1-8km, post 10km vs. pre 1-9km and post 1-3km ($p < 0.05$).*

#### 4.3.4.3 $VCO_2$

Figure 4.9 shows change of $VCO_2$ in pre and post 10km TT. A significant main effect of $VCO_2$ between km number was present ($F_{19, 808.393} = 16.464$, $p < 0.05$). Post hoc indicates that $VCO_2$ is significantly higher in pre 10km vs. pre 1-9km and post 1/2km ($p < 0.05$), post 4km vs. pre and post 1km ($p < 0.05$), post 8km vs. pre 1/6 and post 1km ($p < 0.05$), post 9km vs. pre 1/2/4-8km and post 1km ($p < 0.05$), post 10km vs. pre 1-10km and post 1-9km ($p < 0.05$). In addition, a significant main effect between sex was present ($F_{1, 44.126} = 113.858$, $p < 0.05$); post hoc indicates that male $VCO_2$ is significantly greater than female $VCO_2$ in all pre and post km numbers ($p < 0.05$). No significant main effect between groups was present ($F_{2, 44.128} = 0.873$, $p > 0.05$). No significant interaction of group*sex ($F_{2, 33} = 0.368$, $p > 0.05$) and sex*group*km ($F_{8.673, 2} = 0.765$, $p > 0.05$) was present. A significant interaction of sex*km ($F_{4.337, 1} = 1.605$, $p < 0.05$) was present. Post hoc indicates that male $VCO_2$ data is significantly greater in pre 10km vs. pre 1-9km and post 1-3km ($p < 0.05$), post 8/9km vs. post 1km ($p < 0.05$), post 10 vs. pre 1-9km and post 1-9 ($p < 0.05$); female $VCO_2$ data is significantly greater in pre 10km vs. pre 6/8km ($p < 0.05$), post 9km vs. pre 2/5-8 ($p < 0.05$), post 10km vs. pre 1-10km and post 1-9km ($p < 0.05$). Also, a significant interaction of group*km was present ($F_{8.673, 2} = 1.031$, $p < 0.05$). Post hoc indicates that 15% group $VCO_2$ data is greater in post 10km vs. pre 1-9km and post 1-3/6-8km ($p < 0.05$); 20% group $VCO_2$ data is greater in pre 10km vs. pre 1-9km ($p < 0.05$), post 2/3/9km vs. pre 1/4km ($p < 0.05$), post 4km vs. pre 1/3/4/6km ($p < 0.05$), post 8km vs. pre 1/3/4km ($p < 0.05$), post 10km vs. pre 1-10km and post 1-9km ($p < 0.05$); control group $VCO_2$ data is significantly greater in pre 10km vs. post 1km ($p < 0.05$), post 9km vs. post 1km ($p < 0.05$), post 10km vs. pre 1-9km and post 1-9km ($p < 0.05$).

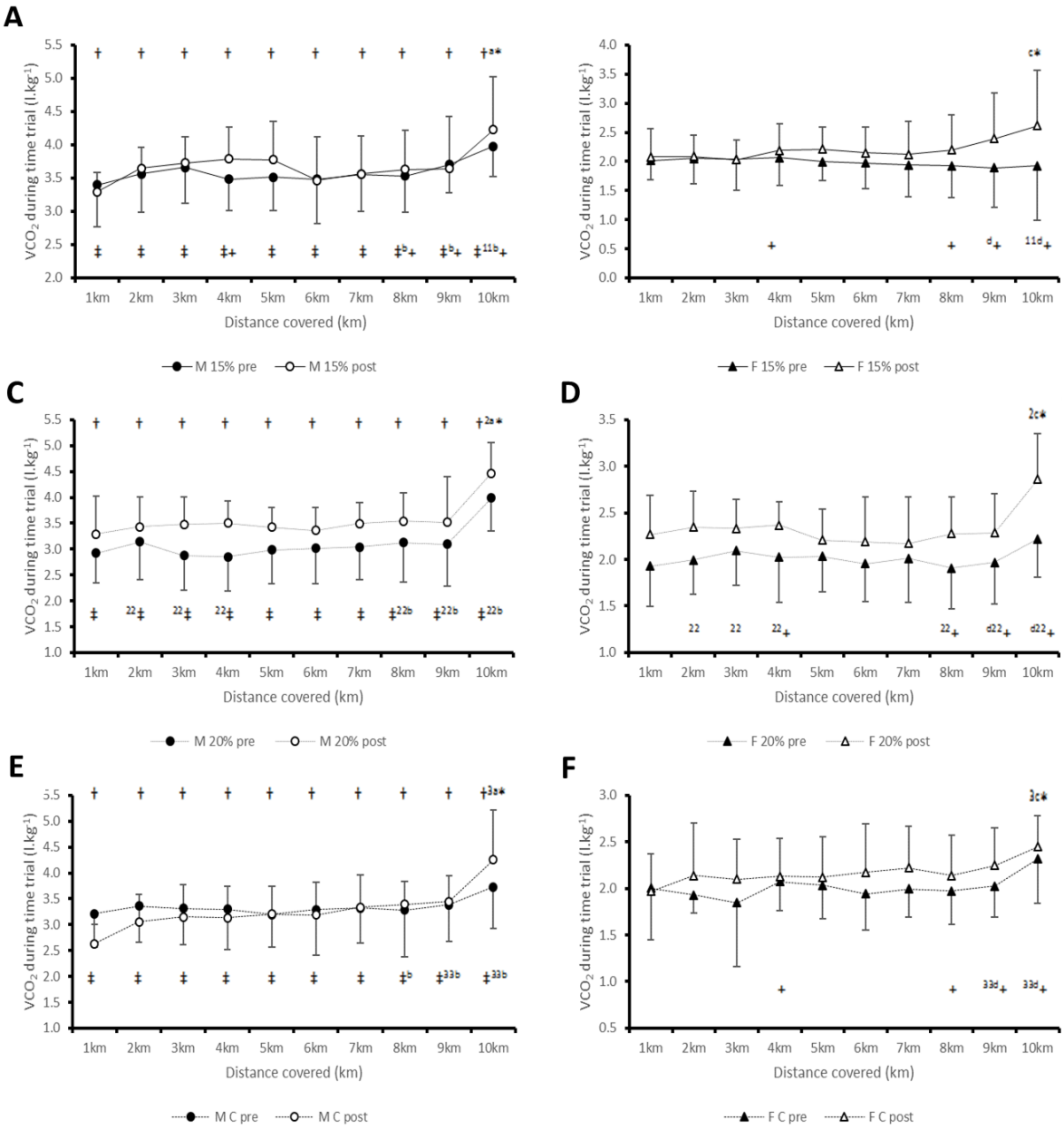


*Figure 4.9: $VCO_2$ rate during 10km TT in pre vs. post testing at each km for 15% males (A), 15% females (B), 20% males (C), 20% females (D), control males (E) and control females (F). * Significantly greater in pre 10km vs. pre 1-9km and post 1/2km ($p < 0.05$). + Significantly greater in post 4km vs. pre and post 1km, post 8km vs. pre 1/6 and post 1km, post 9km vs. pre 1/2/4-8km and post 1km, post 10km vs. pre 1-10km and post 1-9km ($p < 0.05$). † Significantly greater than pre female data ($p < 0.05$). ‡ Significantly greater than post female data ($p < 0.05$). [a] Interaction, male data is significantly greater in pre 10km vs. pre 1-9km and post 1-3km ($p < 0.05$). [b] Interaction, male data is significantly greater in post 8/9km vs. post 1km, post 10 vs. pre 1-9km and post 1-9 ($p <$*

*0.05).* [c] *Interaction, female data is significantly greater in pre 10km vs. pre 6/8km (p < 0.05).* [d] *Interaction, female data is significantly greater in post 9km vs. pre 2/5-8, post 10km vs. pre 1-10km and post 1-9km (p < 0.05).* [11] *Interaction, 15% RR group data is significantly greater in post 10km vs. pre 1-9km and post 1-3/6-8km (p < 0.05).* [2] *Interaction, 20% RR group data is significantly greater in pre 10km vs. pre 1-9km (p < 0.05).* [22] *Interaction, 20% RR group data is significantly greater in post 2/3/9km vs. pre 1/4km, post 4km vs. pre 1/3/4/6km, post 8km vs. pre 1/3/4km, post 10km vs. pre 1-10km and post 1-9km (p < 0.05).* [3] *Interaction, control group data is significantly greater in pre 10km vs. post 1km (p < 0.05).* [33] *Interaction, control group data is significantly greater in post 9km vs. post 1km, post 10km vs. pre 1-9km and post 1-9km (p < 0.05).*

### 4.3.5 Best self-regulated trial, session 1 and 8 normalised sprinting gas measures and heart rate

#### 4.3.5.1 $VO_2$ data

Figure 4.10 shows sprint data, sprint 1 vs. sprint 10 (A-B) and normalised $VO_2$ curve graphs (C-H). A significant main effect of normalised $VO_2$ between best SR trial, session 1 (SES1) and 8 (SES8), sprint 1 (S1) and sprint 10 (S10 (time$^4$)) was present ($F_{5, 145.33} = 28.371$, $p < 0.05$), post hoc indicates that $VO_2$ S10 data is significantly greater than all S1 $VO_2$ data ($p < 0.05$). In addition a significant main effect of sex was present ($F_{1, 29.494} = 50.429$, $p < 0.05$); post hoc indicates that all male $VO_2$ sprint data is significantly greater than all female $VO_2$ sprint data. No significant main effect between groups was present ($F_{1, 29.494} = 1.290$, $p > 0.05$). A significant interaction in time$^4$*sex was present ($F_{5, 145.33} = 4.026$, $p < 0.05$); post hoc indicates that both sexes $VO_2$ S10 data is significantly greater than all their respected $VO_2$ S1 data ($p < 0.05$). No significant interaction in sex*group ($F_{1, 29.494} = 0.87$, $p > 0.05$), group*time$^4$ ($F_{5, 145.33} = 0.721$, $p > 0.05$) and sex*group*time$^4$ ($F_{5, 145.33} = 0.467$, $p < 0.05$).

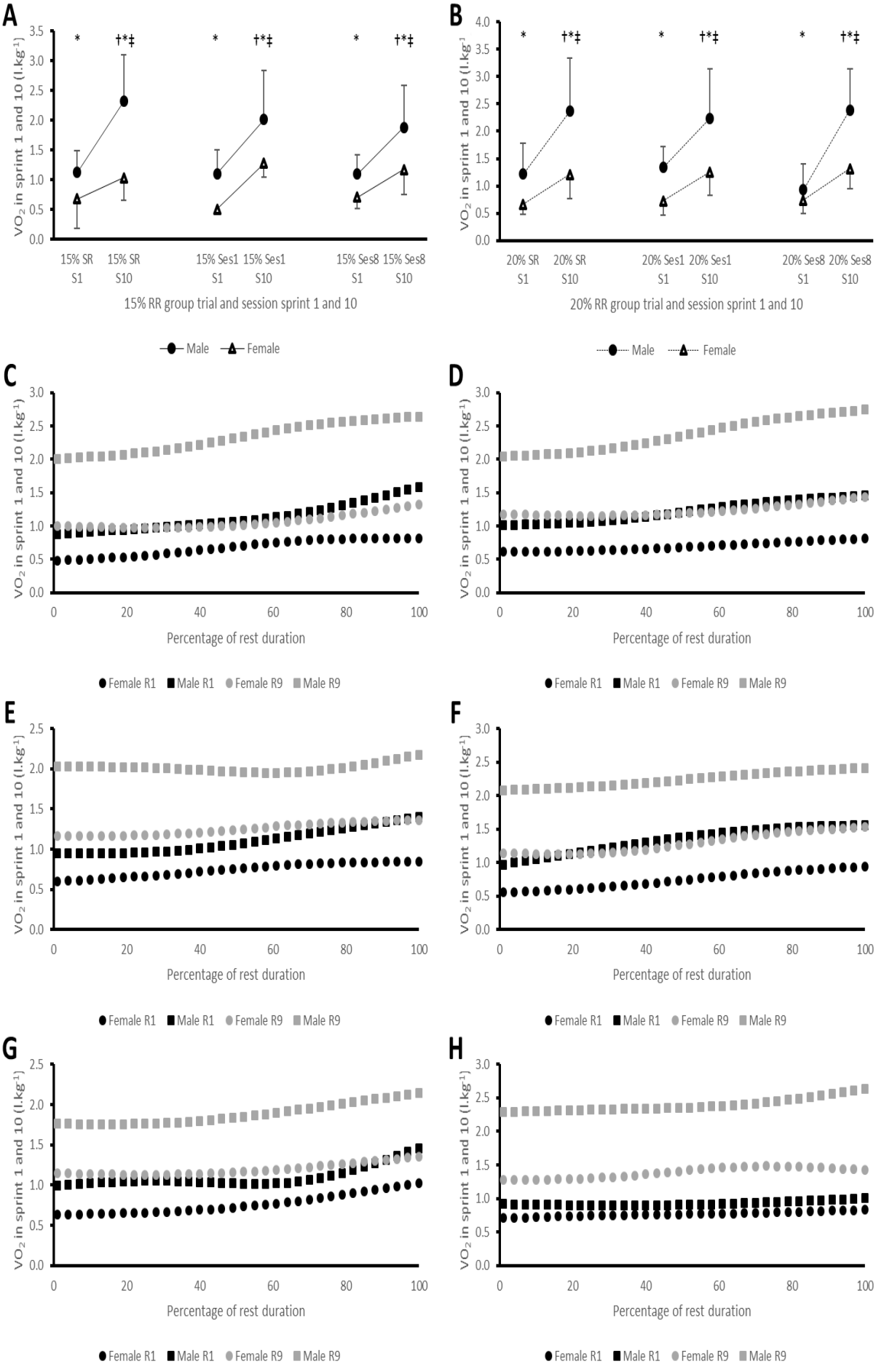
A
B
C
D
E
F
G
H
$VO_2$ in sprint 1 and 10 ($l.kg^{-1}$)
*
†*‡
15% SR S1
15% SR S10
15% Ses1 S1
15% Ses1 S10
15% Ses8 S1
15% Ses8 S10
15% RR group trial and session sprint 1 and 10
20% SR S1
20% SR S10
20% Ses1 S1
20% Ses1 S10
20% Ses8 S1
20% Ses8 S10
20% RR group trial and session sprint 1 and 10
Male
Female
Percentage of rest duration
Female R1
Male R1
Female R9
Male R9

*Figure 4.10: Normalised $VO_2$ in best SR trial, session 1 and 8, data shows sprint 1 vs. sprint 10 in (A) 15% group both sexes, (B) 20% group both sexes, (C) 15% group best SR trial curve both sexes, (D) 20% group best SR trial curve both sexes, (E) 15% group session 1 curve both sexes, (F) 20% group session 1 curve both sexes, (G) 15% group session 8 curve both sexes, and (H) 20% group session 8 curve both sexes. * Significantly greater than females ($p < 0.05$). † Significantly greater than all sprint 1 data ($p < 0.05$). ‡ Interaction, all sprint 10 data is significantly greater than all sprint 1 data in both males and females ($p < 0.05$).*

#### 4.3.5.2 $VCO_2$ data

Figure 4.11 shows sprint data, sprint 1 vs. sprint 10 (A-B) and normalised $VCO_2$ curve graphs (C-H). A significant main effect of normalised $VCO_2$ between time$^4$ was present ($F_{5, 154.12} = 44.905$, $p < 0.05$); post hoc indicates that $VCO_2$ S10 data is significantly greater than all S1 $VCO_2$ data ($p < 0.05$). In addition a significant main effect of sex was present ($F_{1, 31.37} = 32.245$, $p < 0.05$); post hoc indicates that all male $VCO_2$ sprint data is significantly greater than all female $VCO_2$ sprint data. No significant main effect between groups was present ($F_{1, 31.37} = 0.107$, $p > 0.05$). A significant interaction in time$^4$*sex was present ($F_{5, 154.12} = 4.812$, $p < 0.05$); post hoc indicates that both sexes $VCO_2$ S10 data is significantly greater than all their respected $VCO_2$ S1 data ($p < 0.05$). No significant interaction in sex*group ($F_{1, 31.37} = 0.002$, $p > 0.05$), group*time$^4$ ($F_{5, 154.12} = 0.448$, $p > 0.05$) and sex*group*time$^4$ ($F_{5, 154.12} = 0.448$, $p < 0.05$).

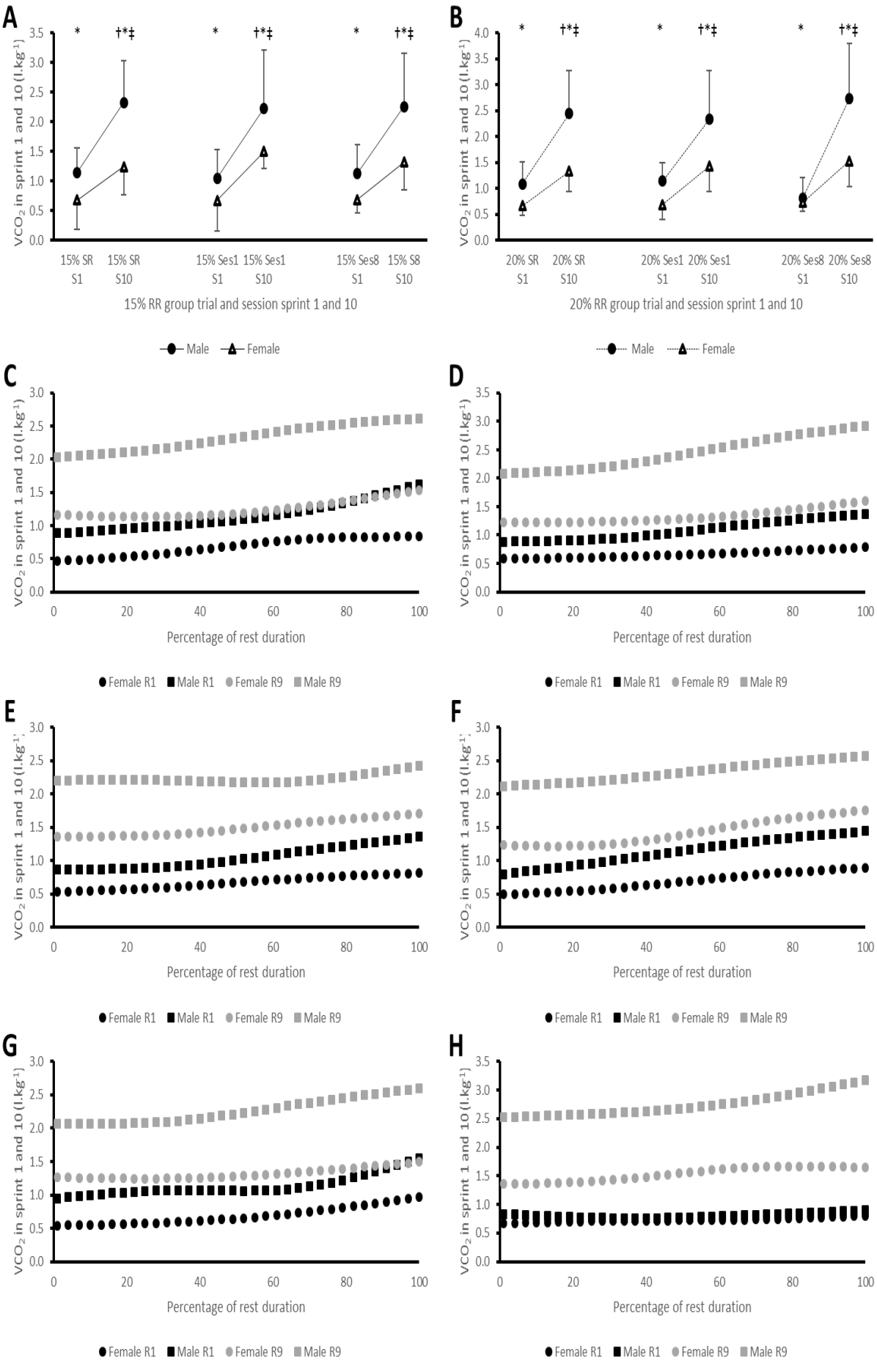
A
B
$VCO_2$ in sprint 1 and 10 (l.kg$^{-1}$)
15% SR S1
15% SR S10
15% Ses1 S1
15% Ses1 S10
15% Ses8 S1
15% S8 S10
15% RR group trial and session sprint 1 and 10
20% SR S1
20% SR S10
20% Ses1 S1
20% Ses1 S10
20% Ses8 S1
20% Ses8 S10
20% RR group trial and session sprint 1 and 10
Male
Female
C
D
E
F
G
H
Percentage of rest duration
Female R1
Male R1
Female R9
Male R9

*Figure 4.11: Normalised $VCO_2$ in best SR trial, session 1 and 8, data shows sprint 1 vs. sprint 10 in (A) 15% group both sexes, (B) 20% group both sexes, (C) 15% group best SR trial curve both sexes, (D) 20% group best SR trial curve both sexes, (E) 15% group session 1 curve both sexes, (F) 20% group session 1 curve both sexes, (G) 15% group session 8 curve both sexes, and (H) 20% group session 8 curve both sexes. * Significantly greater than females ($p < 0.05$). † Significantly greater than all sprint 1 data ($p < 0.05$). ‡ Interaction, all sprint 10 data is significantly greater than all sprint 1 data in both males and females ($p < 0.05$).*

#### 4.3.5.3 Heart rate data

Figure 4.12 shows sprint data, sprint 1 vs. sprint 10 (A-B) and normalised HR curve graphs (C-H). A significant main effect of normalised HR between time$^4$ was present ($F_{5, 148.566} = 27.988$, $p < 0.05$); post hoc indicates that all S10 data is significantly greater than all S1 data ($p < 0.05$). No significant main effect of sex ($F_{1, 32.683} = 0.16$, $p > 0.05$) or group ($F_{1, 32.683} = 1.727$, $p > 0.05$) was present. No significant interaction in sex*time$^4$ ($F_{5, 148.566} = 0.829$, $p > 0.05$), group*time$^4$ ($F_{5, 148.566} = 1.09$, $p > 0.05$), group*sex ($F_{1, 32.683} = 0.003$, $p > 0.05$) and time$^4$*group*sex ($F_{5, 148.566} = 1.078$, $p > 0.05$) was present.

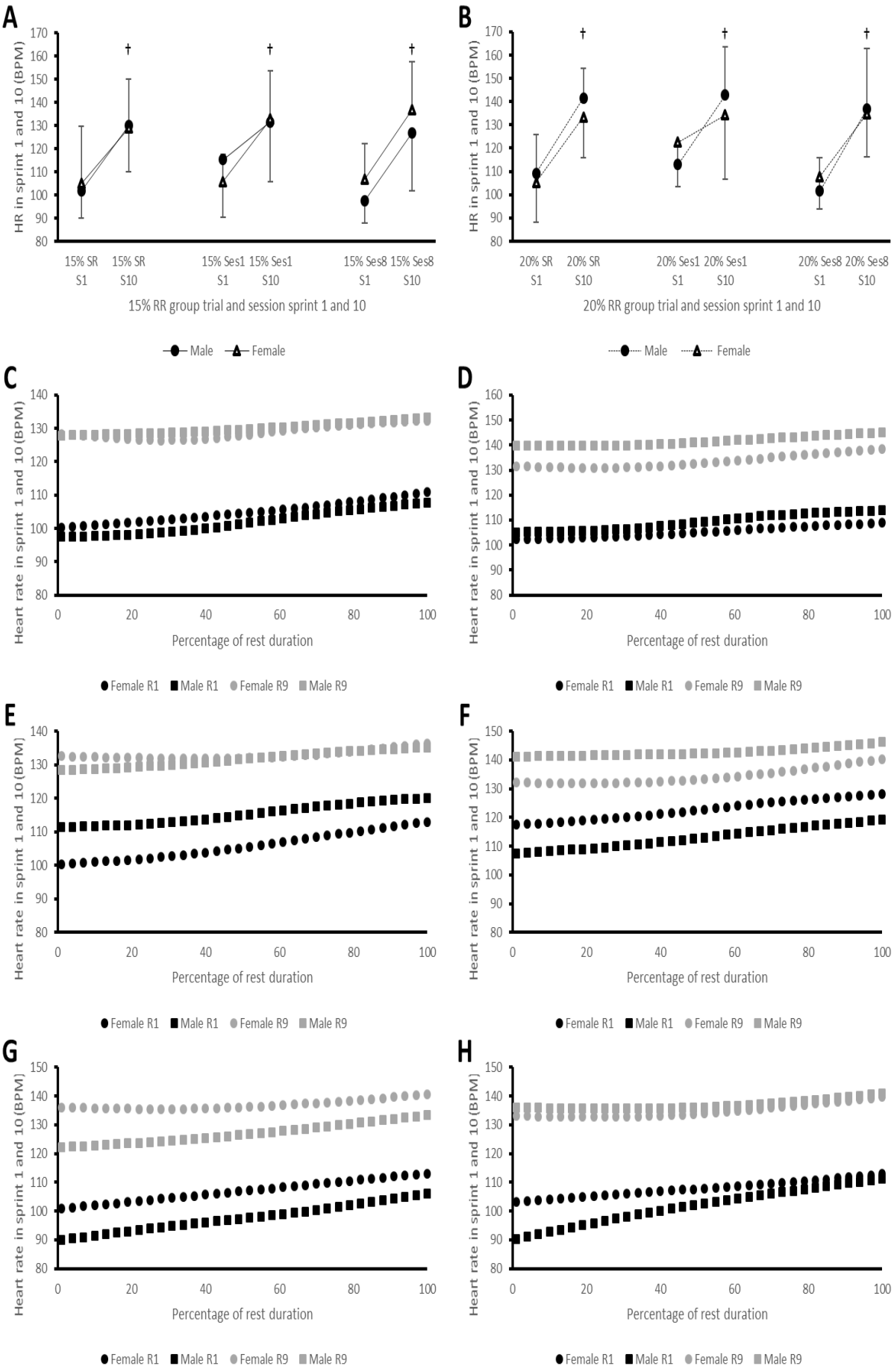
A
HR in sprint 1 and 10 (BPM)
15% SR S1
15% SR S10
15% Ses1 S1
15% Ses1 S10
15% Ses8 S1
15% Ses8 S10
15% RR group trial and session sprint 1 and 10
Male
Female
B
HR in sprint 1 and 10 (BPM)
20% SR S1
20% SR S10
20% Ses1 S1
20% Ses1 S10
20% Ses8 S1
20% Ses8 S10
20% RR group trial and session sprint 1 and 10
Male
Female
C
Heart rate in sprint 1 and 10 (BPM)
Percentage of rest duration
Female R1
Male R1
Female R9
Male R9
D
Heart rate in sprint 1 and 10 (BPM)
Percentage of rest duration
Female R1
Male R1
Female R9
Male R9
E
Heart rate in sprint 1 and 10 (BPM)
Percentage of rest duration
Female R1
Male R1
Female R9
Male R9
F
Heart rate in sprint 1 and 10 (BPM)
Percentage of rest duration
Female R1
Male R1
Female R9
Male R9
G
Heart rate in sprint 1 and 10 (BPM)
Percentage of rest duration
Female R1
Male R1
Female R9
Male R9
H
Heart rate in sprint 1 and 10 (BPM)
Percentage of rest duration
Female R1
Male R1
Female R9
Male R9

*Figure 4.12: Normalised heart rate in best SR trial, session 1 and 8, data shows sprint 1 vs. sprint 10 in (A) 15% group both sexes, (B) 20% group both sexes, (C) 15% group best SR trial curve both sexes, (D) 20% group best SR trial curve both sexes, (E) 15% group session 1 curve both sexes, (F) 20% group session 1 curve both sexes, (G) 15% group session 8 curve both sexes, and (H) 20% group session 8 curve both sexes. † Significantly greater than all sprint 1 data ($p < 0.05$).*

### 4.3.6 Best self-regulated trial, session 1 and 8 normalised resting gas measures and heart rate

#### 4.3.6.1 $VO_2$ data

Figure 4.13 shows resting data, rest 1 vs. rest 9 (A-B) and normalised $VO_2$ curve graphs (C-H). A significant main effect of normalised $VO_2$ between best SR trial, session 1 and 8, rest 1 and rest 9 (time$^5$) was present ($F_{5, 155.026} = 30.167$, $p < 0.05$), post hoc indicates that all $VO_2$ rest 9 data is significantly greater than all rest 1 $VO_2$ data ($p < 0.05$). In addition a significant main effect of sex was present ($F_{1, 31.819} = 51.441$, $p < 0.05$); post hoc indicates that all male $VO_2$ is significantly greater than all female $VO_2$ data. No significant main effect between groups was present ($F_{1, 31.819} = 1.057$, $p > 0.05$). A significant interaction in time$^5$*sex was present ($F_{5, 155.026} = 2.463$, $p < 0.05$). Post hoc indicates that male rest 9 $VO_2$ data is significantly greater than all rest 1 data ($p < 0.05$); female $VO_2$ data is significantly greater in SR rest 9 vs. SR rest 1, SES1 rest 9 vs. SES1 rest 1, and SES8 rest 9 vs. SES8 rest 1 ($p < 0.05$). No significant interaction in sex*group ($F_{1, 31.819} = 0.35$, $p > 0.05$), group*time$^5$ ($F_{5, 155.026} = 0.521$, $p > 0.05$) and sex*group*time$^5$ ($F_{5, 155.026} = 2.169$, $p < 0.05$).

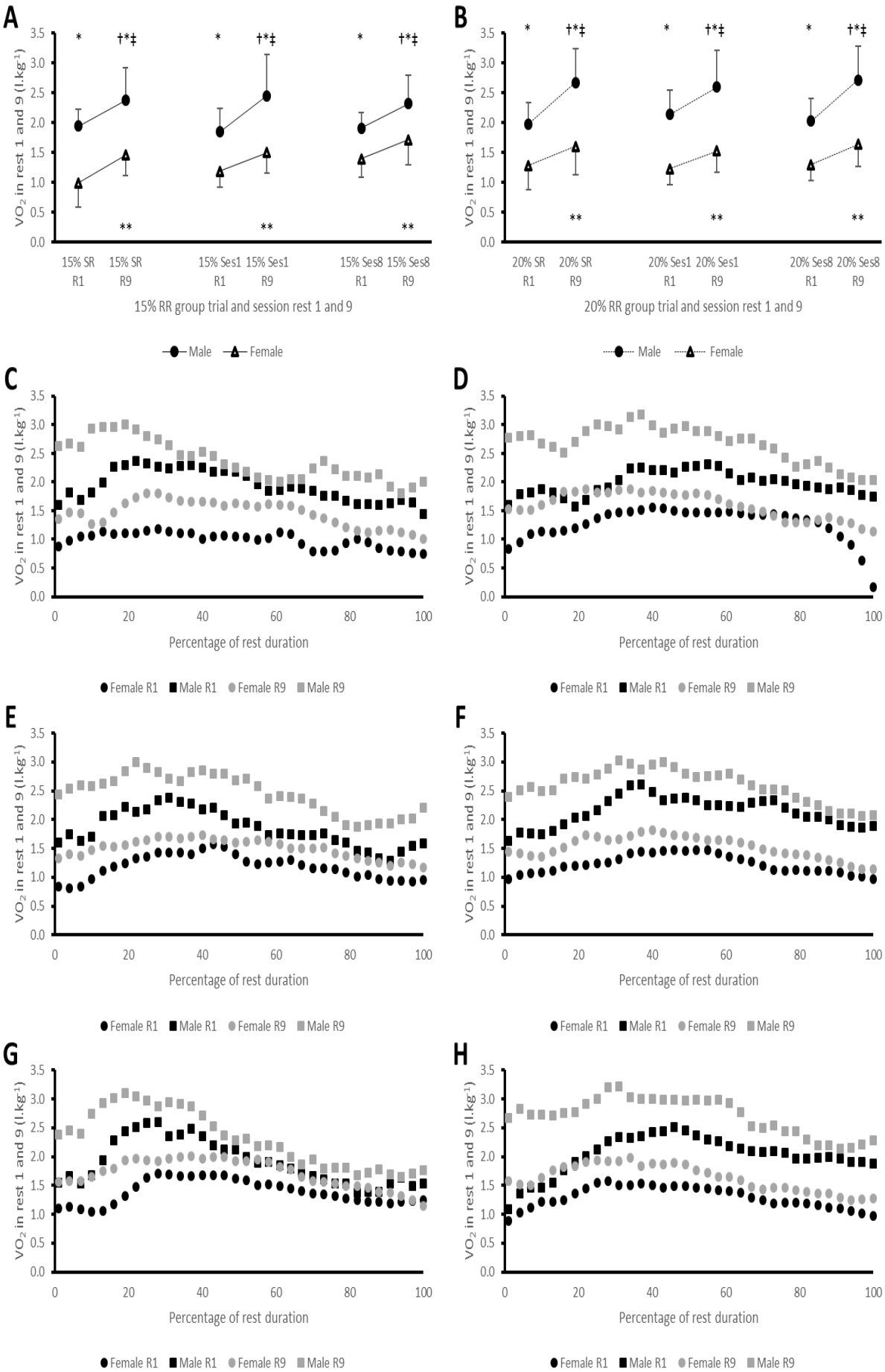
A
B
C
D
E
F
G
H
$VO_2$ in rest 1 and 9 ($l.kg^{-1}$)
15% SR R1
15% SR R9
15% Ses1 R1
15% Ses1 R9
15% Ses8 R1
15% Ses8 R9
15% RR group trial and session rest 1 and 9
20% SR R1
20% SR R9
20% Ses1 R1
20% Ses1 R9
20% Ses8 R1
20% Ses8 R9
20% RR group trial and session rest 1 and 9
Male
Female
Percentage of rest duration
Female R1
Male R1
Female R9
Male R9

*Figure 4.13: Normalised $VO_2$ in best SR trial, session 1 and 8, data shows rest 1 vs. rest 9 in (A) 15% group both sexes, (B) 20% group both sexes, (C) 15% group best SR trial curve both sexes, (D) 20% group best SR trial curve both sexes, (E) 15% group session 1 curve both sexes, (F) 20% group session 1 curve both sexes, (G) 15% group session 8 curve both sexes, and (H) 20% group session 8 curve both sexes. * Significantly greater than females ($p < 0.05$). † Significantly greater than all rest 1 data ($p < 0.05$). ‡ Interaction, all rest 9 data is significantly greater than all rest 1 data in males ($p < 0.05$). ** Interaction, female data is significantly greater in SR rest 9 vs. SR rest 1, SES1 rest 9 vs. SES1 rest 1, and SES8 rest 9 vs. SES8 rest 1 ($p < 0.05$).*

#### 4.3.6.2 $VCO_2$ data

Figure 4.14 shows resting data, rest 1 vs. rest 9 (A-B), normalised $VCO_2$ curve graphs (C-H). A significant main effect of normalised $VCO_2$ between time[5] was present ($F_{5, 155.901} = 30.069$, $p < 0.05$); post hoc indicates that all rest 9 $VCO_2$ data is significantly greater than all rest 1 $VCO_2$ data. In addition, a significant main effect of sex was present ($F_{1, 31.723} = 46.159$, $p < 0.05$); post hoc indicates that all male $VCO_2$ is significantly greater than all female $VCO_2$ data. No significant main effect between groups was present ($F_{1, 31.723} = 0.237$, $p > 0.05$). No significant interaction was present in sex*group ($F_{1, 31.723} = 0.107$, $p > 0.05$), group*time[5] ($F_{5, 155.901} = 0.6$, $p > 0.05$), sex*time[5] ($F_{5, 155.901} = 1.078$, $p > 0.05$). A significant interaction between group*sex*time[5] was present ($F_{5, 155.901} = 2.572$, $p < 0.05$). Post hoc indicates that male 15% $VCO_2$ data is significantly greater in SES1 rest 9 vs. SES1 rest 1 ($p < 0.05$); male 20% $VCO_2$ data is significantly greater in SR rest 9 vs. SR rest 1, SES1 and SES8 rest 9 vs. SES1 and SES8 rest 1 ($p < 0.05$); female 15% $VCO_2$ data is significantly greater in SR rest 9 vs. SR rest 1, SES1 rest 9 vs. SES1 rest 1 ($p < 0.05$); female 20% $VCO_2$ data is significantly greater in SES1 rest 9 vs. SES1 rest 1, and SES8 rest 9 vs. SES8 rest 1 ($p < 0.05$).

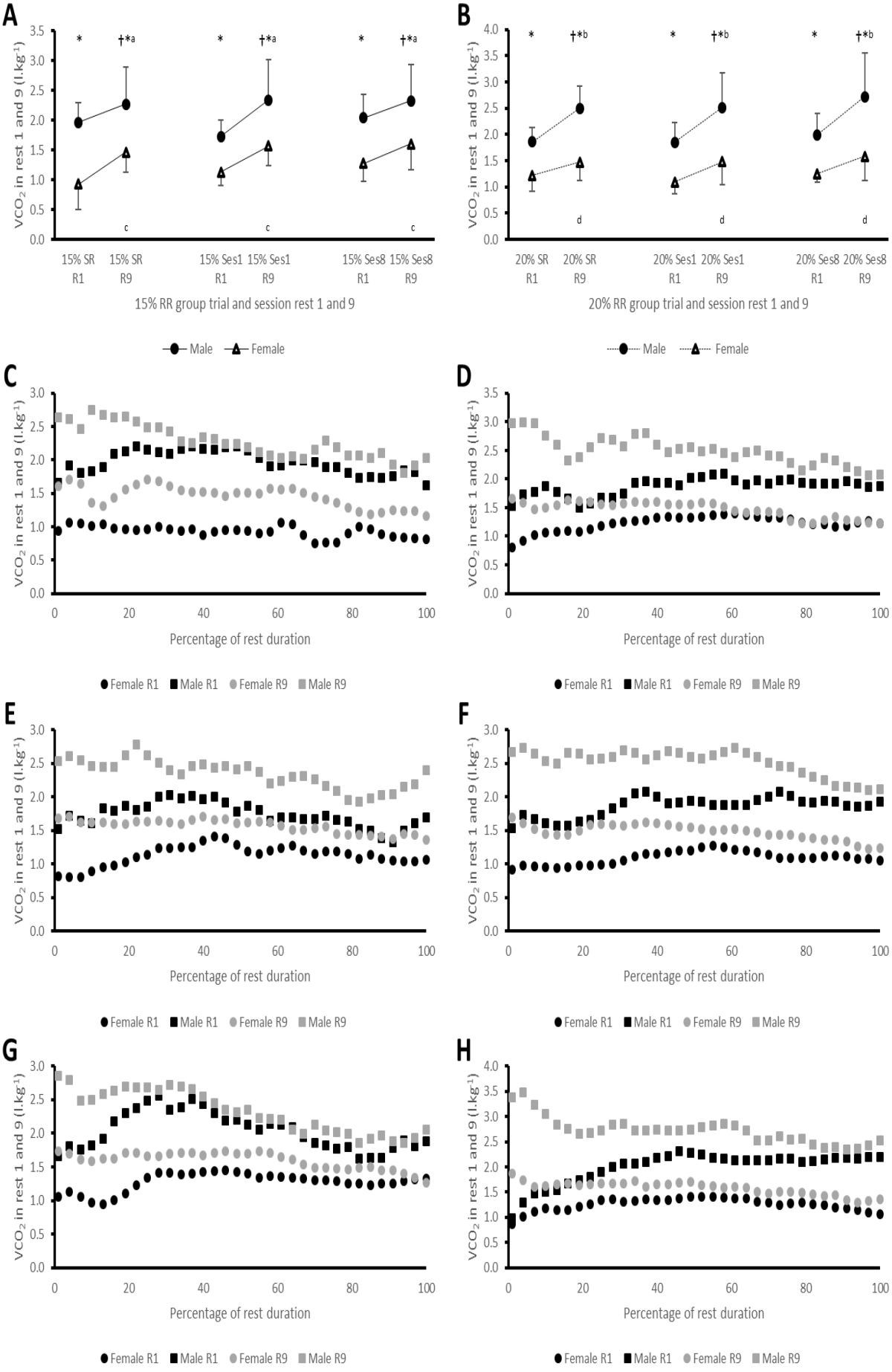
A
B
C
D
E
F
G
H
$VCO_2$ in rest 1 and 9 ($l.kg^{-1}$)
15% SR R1
15% SR R9
15% Ses1 R1
15% Ses1 R9
15% Ses8 R1
15% Ses8 R9
15% RR group trial and session rest 1 and 9
20% SR R1
20% SR R9
20% Ses1 R1
20% Ses1 R9
20% Ses8 R1
20% Ses8 R9
20% RR group trial and session rest 1 and 9
Male
Female
Percentage of rest duration
Female R1
Male R1
Female R9
Male R9

*Figure 4.14: Normalised $VCO_2$ in best SR trial, session 1 and 8, data shows rest 1 vs. rest 9 in (A) 15% group both sexes, (B) 20% group both sexes, (C) 15% group best SR trial curve both sexes, (D) 20% group best SR trial curve both sexes, (E) 15% group session 1 curve both sexes, (F) 20% group session 1 curve both sexes, (G) 15% group session 8 curve both sexes, and (H) 20% group session 8 curve both sexes. * Significantly greater than females ($p < 0.05$). † Significantly greater than all rest 1 data ($p < 0.05$). [a] Interaction, male 15% RR group data is significantly greater in SES1 rest 9 vs. SES1 rest 1 ($p < 0.05$). [b] Interaction, male 20% RR group data is significantly greater in SR rest 9 vs. SR rest 1, SES1 and SES8 rest 9 vs. SES1 and SES8 rest 1 ($p < 0.05$). [c] Interaction, female 15% RR group data is significantly greater in SR rest 9 vs. SR rest 1, and SES1 rest 9 vs. SES1 rest 1 ($p < 0.05$). [d] Interaction, male 20% RR group data is significantly greater in SES1 rest 9 vs. SES1 rest 1, and SES8 rest 9 vs. SES8 rest 1 ($p < 0.05$).*

#### 4.3.6.3 Heart rate data

Figure 4.15 shows resting data, rest 1 vs. rest 9 (A-B) and normalised HR curve graphs (C-H). A significant main effect of normalised HR between time[5] was present ($F_{5, 153.498} = 46.963$, $p < 0.05$); post hoc indicates that all rest 9 data is significantly greater than all rest 1 data ($p < 0.05$). No significant main effect of sex ($F_{1, 31.296} = 0.745$, $p > 0.05$) or group ($F_{1, 31.296} = 0.454$, $p > 0.05$) was present. No significant interaction in sex*time[5] ($F_{5, 153.498} = 0.829$, $p > 0.05$), group*time[5] ($F_{5, 153.498} = 1.09$, $p > 0.05$), group*sex ($F_{1, 31.296} = 0.003$, $p > 0.05$) and time[5]*group*sex ($F_{5, 153.498} = 1.078$, $p > 0.05$) was present.

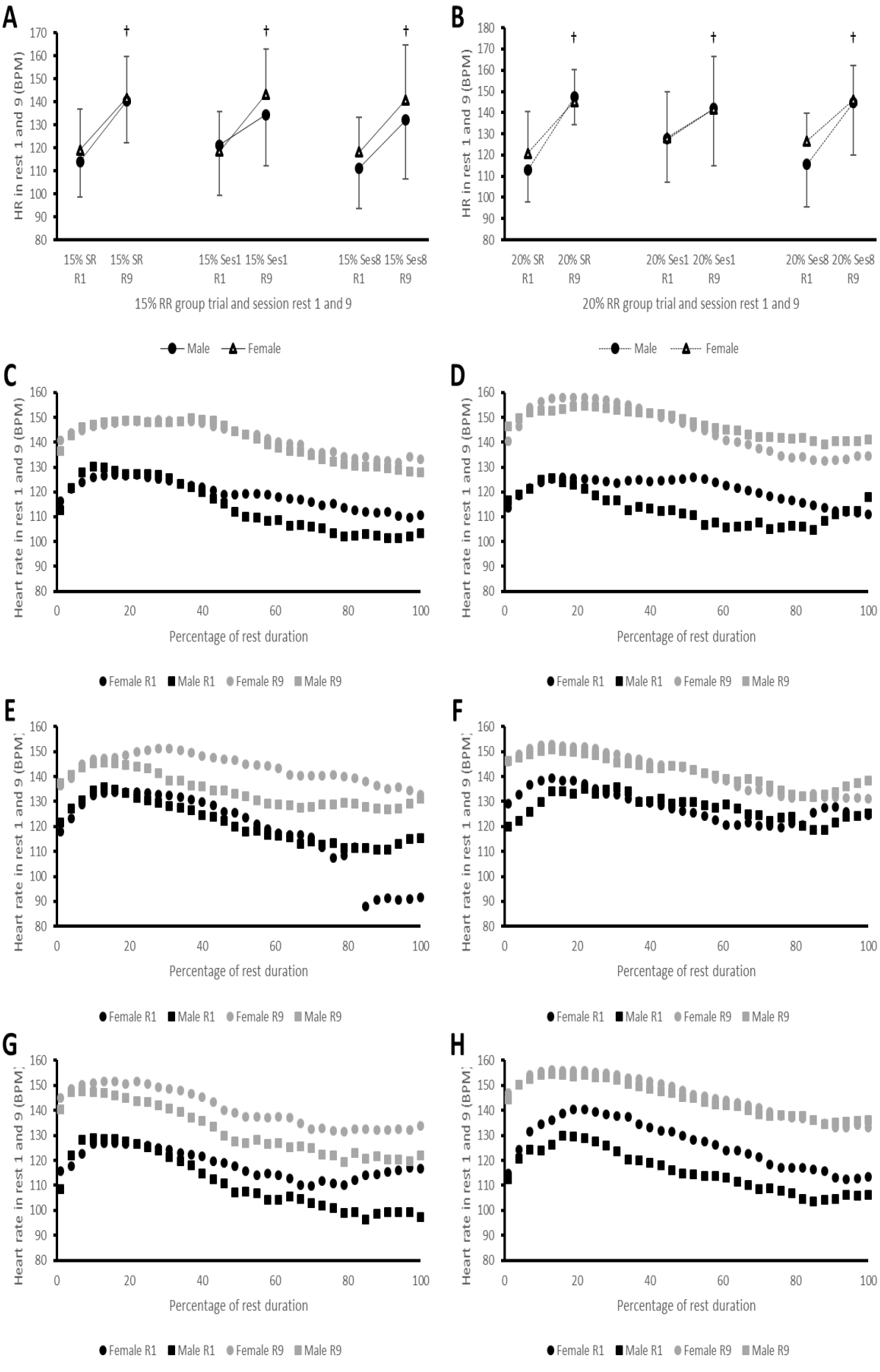
A
HR in rest 1 and 9 (BPM)
15% SR R1
15% SR R9
15% Ses1 R1
15% Ses1 R9
15% Ses8 R1
15% Ses8 R9
15% RR group trial and session rest 1 and 9
B
HR in rest 1 and 9 (BPM)
20% SR R1
20% SR R9
20% Ses1 R1
20% Ses1 R9
20% Ses8 R1
20% Ses8 R9
20% RR group trial and session rest 1 and 9
Male
Female
C
D
E
F
G
H
Heart rate in rest 1 and 9 (BPM)
Percentage of rest duration
Female R1
Male R1
Female R9
Male R9

*Figure 4.15: Normalised heart rate in best SR trial, session 1 and 8, data shows rest 1 vs. rest 9 in (A) 15% group both sexes, (B) 20% group both sexes, (C) 15% group best SR trial curve both sexes, (D) 20% group best SR trial curve both sexes, (E) 15% group session 1 curve both sexes, (F) 20% group session 1 curve both sexes, (G) 15% group session 8 curve both sexes, and (H) 20% group session 8 curve both sexes. † Significantly greater than all rest 1 data ($p < 0.05$).*

### 4.3.7 Gas and heart rate correlations

Table 4.9 shows correlation of sprint and rest $VO_2$, $VCO_2$ and HR between percentage changes of performance measures. Gas and HR measures are overall total of sessions 1 and 8 rest 1 and 9 data (TOTAL). Significant correlation exist in sprint and rest TOTAL $VO_2$ vs. $VO_2$ peak ($r = 0.4$, $p < 0.05$; $r = 0.47$, $p < 0.05$), and TOTAL $VCO_2$ vs. $VO_2$ peak ($r = 0.47$, $p < 0.05$).

Table 5.9: Correlation of sprinting and resting $VO_2$, $VCO_2$, and HR between percentage change of performance measures.

| Measure | TTE | CP | $VO_2$ peak | TT |
|---|---|---|---|---|
| **$VO_2$:** | | | | |
| Sprint TOTAL | r = 0.03 | r = -0.01 | r = 0.4* | r = -0.15 |
| Rest TOTAL | r = 0.03 | r = 0.16 | r = 0.47* | r = -0.04 |
| **$VCO_2$:** | | | | |
| Sprint TOTAL | r = -0.11 | r = 0.06 | r = -0.07 | r = -0.11 |
| Rest TOTAL | r = 0.05 | r = 0.12 | r = 0.47* | r = -0.01 |
| **HR:** | | | | |
| Sprint TOTAL | r = 0.01 | r = -0.06 | r = 0.3 | r = -0.01 |
| Rest TOTAL | r = 0.03 | r = 0.23 | r = 0.06 | r = 0.07 |

** Significant correlation ($p < 0.05$).*

## 4.4 Discussion

It has been suggested that people overestimate the amount of recovery required between sprints (Phillips, Thompson, Oliver., 2014). Therefore, the aim of this study was to determine the effect of reducing self-selected rest by 15 and 20% has on endurance adaptation to repeated sprint training. Following 4 weeks of training endurance adaptations were greater with 15% reduction for $VO_2$ peak (males: 14%, females: -1%) and TTE (males: 6%, females: 8%) when compared to 20% reduction ($VO_2$ peak: males, 9%; females, 0%; TTE: males, 4%; females, 5%). However, endurance adaptations were greater with 20% reduction for CP (males: 8%, females: 10%) and TT (males: -8%, females: -10%) when compared to 15% reduction (CP peak: males, 4%; females, 5%; TT: males, -6%; females, -9%). The magnitude in change for $VO_2$ peak was significantly correlated to TOTAL resting $VO_2$ after sprints ($r = 0.47$, $p < 0.05$) and TOTAL resting $VCO_2$ after sprints ($r = 0.47$, $p < 0.05$) in both sexes.

### 4.4.1 Change in aerobic demand in sprint/ rest 1 vs. sprint 10/ rest 9

A significant increase from sprint 1/ rest 1 vs. sprint 10/ rest 9 for normalised best SR trial in HR, $VO_2$ and $VCO_2$ could indicate a similar trend to Gaitanos et al., (1993) despite the current study's longer rest duration. Gaitanos et al., (1993) reported a dominance in anaerobic ATP production from PCr (80.1%) compared to glycolysis (16.1%) after 10 x 6 sec sprints with a 30 sec recovery. They also discuss that the latter sprints involved a larger aerobic demand and a significant drop in power output. It would appear that a similar drop in glycolysis and increase in PCr use for anaerobic ATP production may have occurred in the current study. Due to the larger aerobic and cardiovascular demand from $VO_2$ and HR in sprint 10/ rest 9 vs. sprint 1 vs. rest 1.

### 4.4.2 Pacing of sprints to maintain mean power output

During the current study, it is thought both males and females have paced their sprints as neither maintained CS performance across trials 1-4 (Table 4.1). This may have occurred as an attempt to protect the body from any homeostatic disturbances (Edwards & Polman., 2013). The significant drop in MPO

compared to the CS (in trials 1-4) may display that participants are pacing their efforts during the 10 trial sprints in order to maintain their MPO. Pacing has previously been found to occur within a single sprint bout, with a 5 sec sprint producing a significantly greater amount of PPO compared to a 15, 30 and 45 sec sprint (Wittekind, Micklewright, Beneke., 2011), despite PPO usually been achieved within the first 0-5 sec of a sprint (Vandewalle, Pérès, Monod., 1987). Similar to female participants in Study 1 and male participants in Study 2, participants in the current study may have paced their efforts as reproducing their CS effort over 10 sprints may have caused homeostatic harm (Edwards & Polman., 2013). This may explain why FI and CV is unaffected in trials 2-4 and trials 3-4 respectively.

### 4.4.3 Self-regulated recovery times between males and females across the 10 sprints

The findings of SR rest duration between males and females (Table 4.1) are consistent with Study 1, with no significant difference between trials or between sexes. Previously it would have been expected that females would have required a shorter SR rest duration vs. males, due to the female's ability to resist peripheral fatigue being greater than males (Laurent et al., 2010; Smith & Billaut 2012). This is now the second study to find that there is no significant difference in SR rest time between sexes. In addition to this, in Study 1, males on average had a shorter SR rest duration compared to females and the opposite has occurred in the current study. Possibly demonstrating that SR rest is a personal selection based off each individual and could be controlled by many factors. Based off unrecorded observations these factors could include willingness to complete the next sprint (Edwards & Polman 2013), comfort, understanding how to maintain MPO (ie: rest for longer), and training level. Despite using self-selected rest, peripheral fatigue linearly increased during the 10 x 6 sec sprints due to rise in HR, $VO_2$ and $VCO_2$ reflecting increasing aerobic contribution, which is consistent with previous research (Bishop., 2012; Goodall et al., 2015; Hureau et al., 2014; Perry et al., 2010; Racinais et al., 2007). This could suggest why the average SR rest duration increases from rest 1 to rest 9 with trial 3 rest 9 been significantly greater than trial 3 rest 1 (Table 4.1)

(Bishop., 2012; Goodall et al., 2015; Hureau et al., 2014; Mendez-Villanueva, Hamer, Bishop., 2008; Perrey et al., 2010; Racinais et al., 2007).

### 4.4.4 $VO_2$ peak test

After eight HIT sessions over a four week period it was found that females saw no increase in $VO_2$ peak (female 15% group: -1%; female 20% group: 0%), whereas males increased by 14% (male 15% group) and 9% (male 20% group) (Figure 4.2). Female $VO_2$ peak results are similar to study 2, which found no improvement for the SR group. It would appear that reducing rest by at least 15% allows improvements in $VO_2$ peak for males but not females. Females may require a reduction in SR of > 20% to see an increase in $VO_2$ peak, to create a larger aerobic response due to a shorter work:rest ratio (Gaitanos et al., 1993; Kavaliauskas, Aspe, Babraj., 2015). This is further suggested by the correlation data within the present study, indicating that increasing aerobic demand during the sprints (TOTAL $VO_2$: $r = 0.4$, $p < 0.05$) and recovery periods (TOTAL $VO_2$: $r = 0.47$, $p < 0.05$) is significantly correlated to the magnitude in change for $VO_2$ peak. Also, significant correlation data between TOTAL rest $VCO_2$ ($r = 0.47$, $p < 0.05$ (Table 4.8)) in $VO_2$ peak percentage change. Indicating that males increased their $VO_2$ peak due to a greater aerobic demand during HIT. Another possibility for females to increase $VO_2$ peak could be increasing the intensity of the HIT by reducing the resistance (< 7.5% body mass (Granata et al., 2016)). Increasing the intensity during HIT (cycling at a greater RPM) is linked with an increase in both mitochondrial respiration and content (Granata et al., 2016; Hughes Ellefsen, Baar., 2017). Females may not have experienced as great an increase in mitochondrial respiration compared to males due to a lack of intensity during HIT (Huges Ellefsen, Baar., 2017). Granata et al., (2016) found that following HIT (4-10 x 30 sec sprints) citrate synthase activity and mitochondrial respiration increased by ~50%. With even short duration sprints (5 sec) during HIT leading to a ~6.8% increase in citrate synthase activity (Linossier et al., 1997). Increasing citrate synthase activity appears to be strongly linked to $VO_2$ max increasing post HIT and endurance training in healthy sedentary or healthy trained young adult males (Vigelso, Andersen, Dela., 2014). There is also the possibility that females do not experience similar

improvements in $VO_2$ peak that men experience following HIT. Research from Vigelso, Andersen, Dela., (2014) compared HIT and endurance training published research from 1983-2013. Vigelso, Andersen, Dela., (2014) speculate that females do not have the same link in improving citrate synthase activity along with $VO_2$ max. An observation was made that research using females only improved citrate synthase activity along with $VO_2$ max when male participants were also used within the same intervention group. However, the data suggesting that females do not see as strong a link in males in improved citrate synthase activity and $VO_2$ max is due to a lack of HIT/ endurance training studies that have used females (Vigelso, Andersen, Dela., 2014). Therefore, based off previous research, females may increase their $VO_2$ peak following HIT by increasing the intensity of their sprints during HIT (Granata et al., 2016; Huges Ellefsen, Baar., 2017; Vigelso, Andersen, Dela., 2014).

Recent work from Kavaliauskas, Steer, Babraj., (2016) found no change in $VO_2$ peak in healthy untrained females post HIT. They also discuss that other research that recruited 6 males and 2 females saw no change in $VO_2$ peak post HIT (Burgomaster et al., 2005). However, other studies increased $VO_2$ peak by ~ 6.9/ ~ 4.7% that have used competitive running female (n = 14) and male (n = 10) participants (Kavaliauskas, Aspe, Babraj., 2015), also by ~ 12/ ~ 13% when using physically active females (n = 7) and males (n = 20) (Yamagashi & Babraj., 2017), and Hazell et al., 2010 increased $VO_2$ max by ~ 9.3/ ~ 9.2/ ~ 3.8% when also using physically active females (n = 13) and males (n = 35). Kavaliauskas, Steer, Babraj., (2016) discuss that training volume may play a key role in improving $VO_2$ peak, specifically longer duration HIT studies (2 sessions a week for 12 weeks) appear to have greater increases in $VO_2$ max compared to short term HIT studies (6 sessions in 2 weeks). Astorino & Schubert., (2014) found a greater response in participants increasing $VO_2$ max (78% of participants) when using a long term HIT protocol (12 weeks) when compared to participants (65%) using a short term protocol (2 weeks). Therefore, it would suggest that the females within the present study may experience an increase in $VO_2$ peak if the duration of the present HIT protocol increased from 4-12 weeks.

Seeking to maintain power output during the training sessions has been suggested to be a potential link to improving power output and endurance measures (Hazell et al., 2010; Lloyd Jones, Morris, Jakeman., 2017; Yamagashi & Babraj., 2017). Hazell et al., (2010) found similar improvements in $VO_2$ max using a 30sec and 10sec sprint and noted that the maintenance of power, through a longer rest, has similar training adaptations to protocols that seek to reduce power output to create a larger aerobic demand by using a shorter work to rest ratio. Yamagashi & Babraj., (2017) record similar PPOs between using a 15sec sprint and 30sec sprint in all sessions. However, they also record a larger significant total work (KJ) in the 30sec group compared to the 15sec group. This indicates that similar training adaptations can be achieved using half the length of a sprint by maintaining PPO and reducing total work (KJ). However, the current study has found that maintenance of MPO through comparing percentage change from the CS to each MPO in the sessions, seeking a lower CV% and FI% is not related to any improvements in $VO_2$ peak, TTE, TT or CP (Table 4.7). Table 4.7 indicates no significant correlation to any measure related to maintenance of power output and percentage change of performance measures. Therefore, maintenance of maximal MPO is not essential for improving $VO_2$ peak.

### 4.4.5 Time to exhaustion test

TTE increased in all training groups and sexes following 4 weeks of HIT (male 15%: ~ 6%, female 15%: ~ 8, male 20%: ~ 4%, female 20%: ~ 5%), regardless of females seeing no increase in $VO_2$ peak. (Figure 4.3). HIT research has found a mixture in TTE percentage increases, Burgomaster et al., (2005) found a ~100% increase in the duration of maintaining 85% $VO_2$ max following 6 HIT sessions over a two week period. This study and others have observed similar percentage increases in incremented TTE after HIT when using competitive male and female runners and triathletes (~ 5%; ~ 6.4/ 4.4/ 1.9% (Jakeman, Adamson, Babraj., 2012; Kavaliauskas, Aspe, Babraj., 2016)). Greater increases appear in untrained healthy females (~ 12% (Kavaliauskas, Steer, Babraj., 2016)), and physically active (3 hours per week) males and females (~ 16/ 13% (Yamagashi & Babraj., 2017)). Improving TTE could be due to

increased muscle citrate synthase activity after HIT, which would increase mitochondrial activity (Burgomaster et al., 2005), increased resting glycogen and PCr stores (Burgomaster et al., 2005; Rodas et al., 2000). Even increasing resting PCr stores decreases an onset of fatiguing mechanisms such as lowering pH (~ 0.5 units), build up in Pi and a reduction in impeded $Ca^{2+}$ dynamics (Balsom et al., 1992; MacLaren & Morton., 2012; Westerblad, Allen, Lannergren., 2002). In addition, Perry et al., (2008) found a ~9% increase in $VO_2$ peak and an increase of ~78% in PCr during the 5 minute stage of a TTE post HIT. PCr and citrate synthase were not measured in the present study. However, given the strong link in resting PCr content and $VO_2$ peak (Kent-Braun & Alexander., 2000), the greater male TOTAL $VO_2$ data been significantly correlated with the increase in $VO_2$ peak, it is possible that the male participants within this study increased their PCr content and citrate synthase activity. This would explain the increase in TTE and $VO_2$ peak in males. The current study found a significant correlation in $VO_2$ peak and TTE ($r = 0.4$, $p < 0.05$). Driller., (2012) found a link between $VO_2$ max and PPO, indicating that when PPO increases during a increment test so does $VO_2$. An increase in PPO would allow a longer TTE, allowing participants to cycle against a greater increment, and therefore an increased $VO_2$ peak (Driller., 2012). However, it has previously been demonstrated that TTE has increased when there is no increase in $VO_2$ peak (Kavaliauskas, Steer, Babraj, 2016; Study 2), even when there was also an increase in PPO post HIT (Study 2).

Jakeman, Adamson, Babraj., (2012) found a significant rightward shift in the lactate curve which resulted in an increase of power by ~30 watts following 6 sessions of 10 x 6 sec cycle sprints. Indicating that both training groups may have increased their TTE by changes in lactate metabolism, which would lead to a greater power output during the test. Post HIT has also shown to increase glycogen in rest and during exercise states, and increase lactate transporter activity $MTC_1$ and $MTC_4$ (Bishop et al., 2008; Burgomaster et al., 2005; Perry et al., 2008). It is thought that $MTC_1$ and $MTC_4$ activity may have increased in the current study due to the substantial rest duration, which would allow time for lactate removal after each sprint (Bishop et al., 2008; Sahlin et al., 1976). Indicating that females may have increased their TTE by an increase in lactate

metabolism, via increased $MTC_1$ and or $MTC_4$ activity, which would lead to a greater power maintenance during the test. With even short duration sprints (5 sec) during HIT leading to a significant increase in the enzyme lactate dehydrogenase (LDH) (Linossier et al., 1993). If LDH activity increased in the present study this would have caused a greater exchange of lactate-pyruvate (Hashimoto, Hussien, Brooks., 2006). As the LDH enzyme is located on the outside of the inner mitochondrial membrane this would allow a greater intracellular lactate shuttle and greater ATP production via oxidation (Hashimoto, Hussien, Brooks., 2006).

Correlation data between tests further suggests that TTE was increased due to an increase in power output (Table 4.8). Increasing TTE is also significantly correlated to decreasing TT ($r = -0.43$, $p < 0.05$), and decreasing TT is significantly correlated to increasing CP ($r = -0.47$, $p < 0.05$). The increase in CP indicates an increase in maximal production rate of oxidative phosphorylation and a higher sustainable work rate (Vanhatalo, Doust, Burnley., 2008). Suggesting that both males and females were able to cycle longer in their TTE test due to an increase in a higher sustainable work rate (Vanhatalo, Doust, Burnley., 2008). The sexes within both training groups appear to experience a similar HR in all measures, with both experiencing a significant increase from rest/ sprint 1 to rest 9/ sprint 10 (Figures 4.12, 4.15). The increase in HR could reflect an increase in aerobic demand, with Yamagishi & Babraj., (2017) finding similar lactate measures along with similar HR measures between their two training groups despite a different of 15 sec sprinting between the groups. Potentially explaining why both sexes increased TTE despite only males increased in $VO_2$ peak.

### 4.4.6 Time trial test

Both training groups and sexes improved their 10km TT test (Figure 4.4) by ~6% (male 15% group), ~8% (male 20% group), ~9% (15% female group), and ~10% (female 20% group). This is consistent with the findings of Jakeman, Adamson, Babraj., (2012), who significantly improved 10km TT performance (~10%), which is similar to using a 30 sec sprint protocol (Burgomaster et al.,

2006; Gibala et al., 2006; Hazell et al., 2010; Kavaliauskas, Steer, Babraj., 2016; Lloyd Jones, Morris. Jakeman., 2017; Yamagashi & Babraj., 2017; Yamagashi & Babraj., 2016). A significant correlation between TT and CP ($r = -0.47$, $p < 0.05$ (Table 4.8)) indicates that TT decreased due to a greater sustainable work rate (Vanhatalo, Doust, Burnley., 2008). This greater sustainable work rate suggests that both sexes and training groups were able to maintain a higher RPM whilst cycling (Driller., 2012). Previous research has shown that with a larger or maintained RPM comes a greater aerobic demand (Marsh & Martin., 1997). The work of Kavaliauskas, Steer, Babraj., (2016) speculate that their improved 10km TT performance could be due to larger oxygen kinetics given the work of Marsh & Martin., (1997) and the no change in $VO_2$ peak found in their female participants. This speculation that TT performance improved due to faster oxygen kinetics is supported by Christensen et al., (2016), who found an increase in oxygen on kinetics following HIT (8-10 x 60 sec sprints separated by 75 sec against ~271 W), with no change in either citrate synthase activity or $VO_2$ peak. The no change in $VO_2$ peak and citrate synthase activity further implies that female participants in the present study possibly did not experience an increase in citrate synthase activity but may have improved oxygen on kinetics leading to greater endurance performance (Jones & Burnley., 2009).

A possible explanation for why females improved their 10km TT performance could be due to increased $MTC_1$ and or $MTC_4$ activity, lactate metabolism, and oxygen kinetics (Christensen et al., 2016; Jakeman, Adamson, Babraj., 2012; Juel et al., 2004; Marsh & Martin., 1997; Pilegaard et al., 1999). Given that increasing TT and TTE performance is significantly correlated ($r = -0.43$, $p < 0.05$) it would suggest that TT performance improved due to similar factors to increasing TTE (Bogdanis et al., 1996; Creer et al., 2004; Ørtenblad et al., 2000). TT in the present study was against a fixed resistance, an increase in sustained work rate would allow participants to pedal faster and therefore decrease their TT (Driller., 2012; Vanhatalo, Doust, Burnley., 2008). The work of Haverty, Kenny, Hodgson., (1988) used trained runners and compared gas analysis data (using $VO_2$ and $VCO_2$) against blood lactate data to establish a link. During long distance running events (5km), participants will eventually

experience a greater aerobic demand to help deal with the energy demands (Haverty, Kenny, Hodgson., 1988). At this point lactate levels plateau and $VO_2$ reaches a steady demand (Haverty, Kenny, Hodgson., 1988). Increasing $VCO_2$ would suggest an increased buffering of lactate by the bicarbonate system (Wasserman et al., 1973). When comparing each km in pre and post testing (eg. $VO_2$/ $VCO_2$/ at pre testing km 1 vs. post testing km 1), $VO_2$ is significantly greater in post testing from 4km – 10km; and $VCO_2$ post 10km (females), 4km and 10km (20% group). Given the increase in $VO_2$ and $VCO_2$, and previous research (Haverty, Kenny, Hodgson., 1988; Linossier et al., 1997; Wasserman et al., 1973), it can be speculated that a greater amount of lactate was present in the post TT vs. pre TT for both sexes and training groups. Possibly indicating an increase in lactate metabolism (Jakeman, Adamson, Babraj., 2012).

### 4.4.7 Critical power test

CP (figure 4.1) also saw an increase in both training groups and sexes (male 15%: ~ 4%; female 15%: ~ 5%; male 20%: ~ 8%; female 20%: ~ 10%), a significant main effect of group and interaction (group*time$^1$) shows that the 20% group improved significantly greater than the control group ($p < 0.05$ (male control: ~ -2%; female control: ~ -6%)). The 15% group percentage increase is close to a significant interaction of group*time$^1$ ($p = 0.062$). Finding a significant increase in CP, using both males and females, post HIT is consistent with recent findings from Kavaliauskas, Steer, Babraj., (2016 (~ 27%)) and Yamagashi & Babraj., (2017 (~ 7.8% and ~ 7.4%)). Interestingly the current study only consisted of 60sec of high intensity work each session, whereas previous research using physically active participants (Yamagashi & Babraj., 2017) involved 60sec – 90sec and 120sec – 180sec of high intensity work. The current study also found a greater percentage increase in the 20% group compared to both of Yamagashi & Babraj., (2017) training groups. Yamagashi & Babraj., (2017) discusses the purpose to using a 15sec sprint is due to the majority of PCr degradation and lactate accumulation occurs within the first 15 seconds of a sprint and has similar effects to a 30sec sprint. The current study could suggest that seeking to maintain MPO when reducing SR by 20% may potentially lead to a greater or similar degradation in PCr and lactate

accumulation when using 10 x 6sec sprints. This may explain the slightly greater percentage increase for the 20% group. The greater percentage increase in CP found Kavaliauskas, Steer, Babraj., (2016) compared to the current study and Yamagashi & Babraj., (2017) could be due to participant choice. Kavaliauskas, Steer, Babraj., (2016) participants took part in the least amount of physical activity in a week (< 3 hours) when compared to the current study (~ 6 hours) and Yamagashi & Babraj., (2017 (≥ 3 hours)). Suggesting that Kavaliauskas, Steer, Babraj., (2016) participants were less when trained, which would potentially allow a greater improvement in CP.

There are potentially sex differences in how the participants have improved the CP. Kavaliauskas, Steer, Babraj., (2016) speculates that their female participants may have improved CP due a greater mitochondrial capacity indirectly measured from maximal citrate synthase activity (Green et al., 1999). Thought to occur given the research that has found increase in citrate synthase activity post HIT (Burgomaster et al., 2005; Linossier et al., 1993). However, as discussed earlier, there is potential that the female participants within the current study may not have increased their citrate synthase activity given the no change in $VO_2$ peak (Vigelso, Andersen, Dela., 2014). Instead, females may have seen an improvement in CP due to increased lactate transporter activity which have both been found to increase post HIT (Burgomaster et al 2008; Perry et al., 2008; Rodas et al., 2000). This would allow the resistance of fatigue from an accumulation of Pi and lead to a greater sustained power and velocity during the CP test (Jones et al., 2010; Westerblad, Allen, Lannergren., 2002).

### 4.4.8 Limitations

The current study did not take into account what sport or activity the participants partake in. The males within the control group may have entered an intense training phase of their normal training due to their sport/activity, possibly explaining their ~ 6% increase in TTE test. Dietary intake before each trial/ session/ test was not strictly monitored, the mood state of the participants may have had an affect when self-regulating their own rest periods (Adan., 1994; Davis., 1995). It has been shown that stimulants high in caffeine, such as

coffee, tea and cola, reduce the release of serotonin that cause lethargic states (Davis., 1995). It is also established that humans take these stimulants to self-regulate their energy level in normal work schedules (Adan., 1994). Participants who regularly take stimulants could possibly gain an advantage over participants that don't during SR HIT.

### 4.4.9 Conclusion

For the first time, this study has demonstrated that reducing self-selected rest times by 15% and 20%, during repetitive short sprint duration HIT, leads to increased performance adaptions in $VO_2$ peak (males), TTE, 10km TT and CP. This study has also demonstrated that creating specific rest times for each participant has similar effects to using work to rest ratios that are designed for improving endurance performance measures. Further research should identify if reducing female self-selected rest duration by > 20% or reducing the resistance of the sprint (< 7.5% body mass) leads to an improvement in $VO_2$ peak. Further research should also seek to identify why a participant has selected a rest period and why they deem it sufficient to maintain maximal MPO. This may aid practitioners in using self-regulated rest as a training tool.

### 4.4.10 Practical implications

The present study has identified four major findings. 1) Reducing SR rest by 20% leads to greater improvements in performance measures compared to 15% reduction and still have an increase in $VO_2$ peak of ~8.9% in males. 2) Using an extra two HIT sessions (eight sessions) and reducing SR rest during HIT leads to greater and significant increases in endurance measures when compared to not reducing SR over six HIT sessions (Study 2). 3) Maintenance of CS MPO during the HIT sessions doesn't appear to be a major factor for increasing endurance measures, which is in contrast to the proposed importance given by others (Hazell et al., 2010; Lloyd Jones, Morris, Jakeman., 2017). 4) Further evidence that a 30 sec sprint during HIT is not required and that the early part of a 30 sec sprint is a major factor for improving performance measures (Jakeman, Adamson, Babraj., 2012). From this information

practitioners should understand that reducing SR by 20% leads to an individual response during HIT.

# 5 Chapter 5 - General discussion

## 5.1 Introduction

The key aims of this PhD thesis was to identify the effects of using self-regulated (SR) rest in relation to maintaining mean power output (MPO) between sexes and its potential use during high intensity training (HIT) to create positive performance adaptations. Within HIT research it has been previously speculated that the maintenance of power during HIT is a main factor for increasing performance measures (Hazell et al., 2010; Lloyd Jones, Morris, Jakeman., 2017; Yamagishi & Babraj., 2017). Yet little research is available to indicate if maintenance of power output during HIT is an important factor for performance adaptation. Traditionally HIT research involves a decrement in power output during HIT to create a greater aerobic response during the sprints (Bogdanis et al., 1996; Gaitanos et al., 1993), which is thought increase endurance measures such as $VO_2$ max (Sloth et al., 2013). These studies have used 30 sec sprints to create a greater aerobic response, with the last 15 sec of a 30 sec sprint been predominantly aerobic (> 50% (Parolin et al., 1999)). However, there is emerging research that indicates that the duration of the sprint is not vital, with durations of 6-15 sec finding similar adaptations to that of a 30 sec sprint protocol (Hazell et al., 2010; Kavaliauskas, Aspe, Babraj., 2015; Kavaliauskas, Steer, Babraj., 2016; Jakeman, Adamson, Babraj., 2012; Lloyd Jones, Morris, Jakeman., 2017; Yamagishi & Babraj., 2017). If maintenance of power is a main factor for increasing performance measures then allowing participants to SR their recovery to maintain MPO may also lead to an improvement in $VO_2$ peak, time to exhaustion, time trial, and power output. Therefore, using SR rest within HIT would create specific work to rest ratios for each individual participant. There is also debate regarding the resistance applied to the sprint during HIT, with 7.5% body mass (BM) traditionally used (Table 1.1 and 1.2), and emerging research indicating that females should adopt a lower resistance (6.5-7% BM (Kavaliauskas, Steer, Babraj., 2016; Yamagishi & Babraj., 2017)). This is due to morphological differences between sexes such as greater fat mass in females and greater muscle mass in males (Perez-Gomez., 2008). Lowering the resistance will allow the intensity of the HIT to increase (i.e. a greater pedal frequency during the sprints) which may

reflect into greater power outputs for females (Billaut & Bishop., 2009). This thesis will identify sex differences in maintaining MPO and magnitude in change in performance measures using SR rest and 7.5% BM resistance.

## 5.2 Summary of main findings

### 5.2.1 Self-regulated recovery

In Studies 1-3 there was no significant difference in trial average SR rest duration between SR trials and between sexes (Studies 1 and 3 (Tables 3.1 and 5.1)). In Study 1 it was hypothesised that females would require a shorter SR rest duration potentially due to differences in percentage area of skeletal muscle fibre type between males and females (Glenmark, Hedberg, Jansson., 1992; Hicks, Kent-Braun, Ditor., 2001). Females have been found to have a significantly greater percentage area of type I muscle fibres with males showing a significantly greater percentage area of type IIa muscle fibres in the vastus lateralis (Roepstorff et al., 2006; Staron et al., 2000). The greater recruitment of type I muscle fibres leads to a greater fat oxidation capacity (Roepstorff et al., 2006; Staron et al., 2000), which is associated with developing less peripheral fatigue compared to a greater recruitment of type IIa muscle fibres which leads to higher glycolytic enzyme activity (Roepstorff et al., 2006; Russ et al., 2005). Repeat sprint activity research has shown that males are able to produce greater absolute power outputs then females but they also see a greater decrement in power output when compared to females (Billaut et al., 2011; Laurent et al., 2010). This could be due to females having the capacity to recover phosphocreatine (PCr) at a greater rate compared to males (Kent-Braun & Alexander., 2000), which would aid repeat sprint ability performance (Gaitanos et al., 1993; Rodas et al., 2000). Therefore, it was thought that females would self-select a shorter SR rest duration when compared to males. However, Studies 1 and 3 have consistently found no significant difference between sexes in duration of SR rest, and found inconsistencies in average SR rest duration between sexes, with males demonstrating a shorter SR rest duration in Study 1 and vice versa in Study 3. The similar SR rest duration between sexes could be due to the high intensity nature of the study (Knechtle

et al., 2004). Sex peripheral fatiguing differences (males: greater carbohydrate oxidation, females: greater fat oxidation) appear to diminish the closer participants exercise to their maximal intensity (55 vs. 75% of $VO_2$ peak (Knechtle et al., 2004)). Therefore, during this thesis participants would may have exercised at ≥ 75% of their $VO_2$ peak during sprints. Potentially explaining the similar duration in SR rest duration despite females been viewed as more fatigue resistant than males (Billaut & Bishop., 2009).

It is not firmly understood as to what regulates self-selected rest periods and what indicates to a participant that they have rested adequately to repeat another sprint bout that replicates their criterion sprint MPO effort. It is understood that exercise induced skeletal muscle fatigue is caused by peripheral (depletion in glycogen and PCr) and central fatigue (decreased firing rate (Amann., 2011; Amann., 2012; Amann & Dempsey., 2008; Decorte et al., 2012; Froyd et al 2016; Froyd, Millet, Noakes., 2013; Kent-Braun., 1999; Mendez-Villanueva et al., 2012)). This research shows that during maximal intensity exercise and self-paced cycling that peripheral fatigue occurs and progressively increases as the number of completed bouts or duration of exercise increases. An increase in peripheral fatigue during Studies 1-3 is suggested by the significant increase in $VO_2$, $VCO_2$ and heart rate (HR) from rest 1/ sprint 1 to rest 9/ sprint 10 (Figure 2.1-3, 3.4-5, and 4.10-15). These increases in $VO_2$, $VCO_2$ and HR suggest an increase in peripheral fatigue due to greater use of peripheral organs (heart and lungs), which also leads to an increase in oxygen delivery to ensure that skeletal muscle can still contract at a high rate (Marcora., 2009; Zając et al., 2015). With an increase in aerobic demand during repeat sprint activity demonstrating a decrease power output, PCr availability and inhibition of glycolysis (Bogdanis et al., 1996; Gaitanos et al., 1993; Parolin et al., 1999). Once peripheral fatigue has already been firmly established central fatigue is thought to occur and overall only accounts for ~20% of fatigue after maximal voluntary contractions (Kent-Braun., 1999). The perception of effort is thought to come from peripheral mechanisms known as afferent feedback, which senses effort from the heart, lungs and from muscle spindles and Golgi tendon organs (Marcora., 2009). Participants within the present studies could be self-regulating their rest until this sense of effort has

reduced, which might be decisive in a participant deciding when they wish to begin their next sprint. Study 3 shows that SR rest duration on average is greater in rest 9 compared to rest 1 and rest 9 is significantly greater in rest 9 vs. rest 1 in trial 3 for both males and females (Table 4.1). With HR, $VO_2$ and $VCO_2$ significantly increasing it suggests that rest 9 SR rest duration increases due to a greater sense of effort (Marcora., 2009), and or based off previous research it could suggest that central fatigue became established by rest 9 (Kent-Braun., 1999).

Study 1 is consistent with previous research that participants who can successfully maintain their MPO are over estimating their SR recovery by at least 10% (Phillips, Thompson, Oliver., 2014). However, when average SR recovery was reduced by 15% in Study 1 it was found that MPO was significantly less when compared to the criterion sprint, more so in females (~9.8% decrease) than males (~1.2% decrease (Table 2.1)). The significant improvements in endurance measures in Study 3 (HIT with reduced SR rest of 15-20% (Figure 4.1-4)) vs. the non-significant increase in endurance measures in Study 2 (HIT with SR rest (Figure 3.1)) could indicate that the 10% over-estimation in SR recovery effects HIT negatively. The findings of Yamagishi & Babraj., (2017) indicate a plateau in $VO_2$ peak changes after six sessions, which indicates that the use of an extra two HIT sessions in Study 3 compared to Study 2 is not a factor for the magnitude in change differences between the two studies. If the greater improvement in endurance measures is caused by a reduction in SR rest (15-20%) then seeking to further identify at what point a participant is able to reproduce their maximal MPO during SR rest might be vital for obtaining the greatest magnitude in change in endurance measures.

### 5.2.2 Self-regulated recovery and reduced self-regulated rest vs. fixed rest on performance adaptations

Traditionally HIT research uses fixed rest periods between sprints in relation to the sprint duration to create a work to rest ratio, with a 1:8 work to rest ratio been commonly used (Tables 1.1 and 1.2). Study 2 has consistencies with the work of Kavaliauskas, Aspse, Babraj., (2015 (6 x 10 sec sprints, 7.5% BM

resistance)) who found that using specific work to rest ratios (1:3, 1:8, 1:12) leads to specific performance outcomes. With shorter work to rest ratios finding greater improvements in $VO_2$ peak (1:3, ~6.9%; 1:8, ~4.7%; 1:12, ~0.3%), time to exhaustion (TTE (1:3, ~6.3%; 1:8, ~4.4%; 1:12, ~1.9%)) and time trial (1:3, ~3.1%; 1:8, ~2.4%; 1:12, ~2.4%). Whereas longer work to rest ratios found greater improvements in Wingate peak power output (PPO (1:3, ~4.3%; 1:8, ~7.1%; 1:12, ~8.5%)) and Wingate MPO (1:3, ~0.3%; 1:8, ~4.6%; 1:12, ~5.3%). The fixed rest (FR (30 sec)) training group in Study 2 found only an improvement in $VO_2$ peak (~5%), which is consistent with Kavaliauskas, Aspse, Babraj., (2015) who speculate that endurance testing increased due to a greater aerobic response during HIT due to a short work to rest ratio. Similar to Kavaliauskas, Aspse, Babraj., (2015), the SR group (~1:17) experienced greater improvements in Wingate PPO (SR: 1.1%; FR: -4%) and Wingate MPO (SR: 0.4%; FR: -2.1%). It is thought that the improvement in power output in the SR group is linked with a greater improvement in TTE (SR: ~3%; FR: ~0%) and TT (SR: ~-3%; FR: ~3%) compared to the FR group (Bulbulian, Wilcox, Darbos., 1986; Noakes., 1988). Increasing Wingate PPO or MPO may not be strongly correlated to increasing TTE (PPO: r = 0.05; MPO: r = 0.03) or TT (PPO: r = -0.26; MPO: r = -0.31) but a moderate correlation indicates a link between improving TTE is linked with improving TT (r = -0.41). Given that TT performance can be increased due to an increased sustained work rate due to an increased power output (Driller., 2012) and TTE has been found to increase through an increase in power (Jakeman, Adamson, Babraj., 2012), it is thought that this moderate correlation is a reflection of the SR group's increase in power output.

In Study 3 participants completed eight sessions of HIT (10 x 6 sec sprints 7.5% BM resistance) but with a reduction in SR rest of 15 and 20% due to the known 10% over-estimation in required recovery (Phillips, Thompson, Oliver., 2014; Study 1). These reductions led specific work to rest ratios for each individual participant, with average work to rest ratios of ~1:16.3 (male 15% (M15)), ~1:12.2 (female 15% (F15)), ~1:12 (male 20% (M20)) and ~1:12.8 (female 20% (F20)). In contrast to Kavaliauskas, Aspse, Babraj., (2015), both training groups and sexes in Study 3 found improvements in TTE (M15: ~6.4%; F15: ~7.6%;

M20: ~4.1%; F20: ~5%), TT (M15: ~6%; F15: ~8.7%; M20: ~8%; F20: ~10.4%) and critical power (M15: ~4.1%; F15: ~5%; M20: ~8.2%; F20: ~9.6%), with only the male training groups finding an improvement in $VO_2$ peak (M15: ~13.8%; F15: ~-0.6%; M20: ~8.9%; F20: ~0%). Hazell et al., (2010) speculates that the reproduction of power output during HIT is a key factor for improving performance measures, and found improvements in performance measures when using 1:8 (30 sec sprint), 1:12 (10 sec sprint) and 1:24 (10 sec sprint) work to rest ratios. The 1:24 work to rest ratio found greater improvements than 1:12 work to rest ratio in $VO_2$ max (1:12, ~3.8%; 1:24, ~9.2%) TT (1:12, ~3%; 1:24, ~3.5%), Wingate PPO (1:12, ~4.2%; 1:24, ~8.5%) and Wingate MPO (1:12, ~2.9%; 1:24, ~6.5%). The 1:12 and 1:24 training groups experienced a significantly greater reproducibility of training PPO, MPO and minimum power output compared to the 1:8 group, with the 1:24 group producing slightly greater power reproducibility than the 1:12. From this data Hazell et al., (2010) speculates that the increased rest duration in the 1:24 group led to greater improvements than the 1:12 group and some similar improvements to the 1:8 group ($VO_2$ max: ~9.3%; TT: ~5.2%; PPO: ~9.5%; MPO: ~12.1%). Study 1 found a significant correlation with maintaining trial MPO and increasing $VO_2$ and $VCO_2$ response ($r = 0.78$ and $r = 0.73$) respectively (Tables 2.3-5). Suggesting that a longer rest duration to allow greater reproducibility of MPO is potentially linked with improving endurance measures, given that a greater aerobic response during HIT is thought to increase these endurance measures (Bogdanis et al., 1996; Gaitanos et al., 1993; Kavaliauskas, Aspse, Babraj., 2015).

### 5.2.3 Maintenance of power output

The use of self-regulated rest and the effects of maintaining MPO appear to be inconsistent between Studies 1-3. In Study 1 it was demonstrated that males could maintain their criterion sprint (CS) MPO greater than females (Table 2.1). The SR group in Study 2 were unable to maintain their CS MPO as well as the males in Study 1 (Tables 3.1). In Study 3 both males and females were unable to maintain their CS MPO by a similar amount (Table 4.1). This could indicate that the ability to maintain MPO through SR rest is different for each individual,

possibly explaining why these three studies found multiple participants that could not maintain their MPO (n = 11) in two out of four trials. This individual difference might explain why Phillips, Thompson, Oliver., (2014) and Glaister et al., (2010) found that all their participants could maintain MPO or sprint speed respectively across all trials. What appears to be consistent between the three studies and the work of Hopkins, Schabort, Hawley (2001) is that participants are able to maintain their MPO more effectively (lower coefficient of variation between the 10 x 6 sec sprints MPO) in the latter trials compared to the earlier trials. Study 1 found that a greater sum of MPO during the trials is significantly correlated to increasing $VO_2$ (r = 0.78) and $VCO_2$ (r = 0.73) but not HR (r = 0.3). Whilst $VO_2$, $VCO_2$ and HR was significantly greater in sprint 10/ rest 9 vs. sprint 1/ rest 1, male $VO_2$ and $VCO_2$ data was significantly greater then female data at all measured points (Studies 1-3). This potentially explains why male training group participants in Study 3 saw significant increases in $VO_2$ peak whereas females saw no change (Figure 4.2). Given that increasing aerobic demand during HIT is thought to be a key factor for increasing endurance measures (Bogdanis et al., 1996; Gaitanos et al., 1993; Kavaliauskas, Aspse, Babraj., 2015; Sloth et al., 2013). The significant increase in $VO_2$, $VCO_2$ and HR from sprint 1/ rest 1 to sprint 10/ rest 9 could indicate that despite using SR rest (Study 1: ~100 sec; Study 2: ~104 sec; Study 3: ~94 sec), seeking to maintain MPO could lead to similar adenosine triphosphate (ATP) turnover changes from PCr and glycolysis as found by Gaitanos et al., (1993). Gaitanos et al., (1993) conducted 10 x 6 sec (7.5% BM resistance) cycle sprints separated by a recovery (30 sec) that was deemed insufficient for optimal recovery to reproduce maximal power output. They found that PPO and MPO significantly declined by 15.9% and 12.6% after sprint 5, and by 33.4% and 26.6% after sprint 10 respectively. They also reported that ATP turnover from PCr increased from sprint 1 (49.6%) to sprint 10 (80.1%), ATP turnover from glycolysis decreased from sprint 1 (44.1%) to sprint 10 (16.1%), and suggest that aerobic metabolism increased significantly from sprint 1 to sprint 10 with an total ATP turnover of 13.1 mmol. kg dry $wt^{-1}$. $s^{-1}$. This may suggest that maintaining MPO leads to similar metabolic demand then seeking to decrement power output during HIT, and explain why endurance measures improved in Studies 2-3. However, Studies 2-3 found no significant correlation between maintaining CS

MPO during trials/ sessions and performance measures (Tables 3.2 and 4.7). A negative moderate correlation in Study 2 indicates that seeking to decrement MPO is linked to increasing $VO_2$ peak (Table 3.2). Therefore, correlation data in Studies 2-3 indicates that the speculative suggestion from Hazell et al., (2010) and Lloyd Jones, Morris, Jakeman., (2017), that maintenance of power output could be a key factor for improving performance measures, is incorrect. However, when giving participants the instruction to maintain their MPO during HIT causes an increase in $VO_2$ peak (males), TTE, TT, and CP from pre to post testing measures regardless of the participants successfully or unsuccessfully maintaining their CS MPO.

### 5.2.4 Sex specific improvements in endurance following reduced self-regulated high intensity training

Despite no significant difference between the second CS and session MPO for both sexes and training groups in Study 3, the results from Study 3 possibly indicate that the reduced SR rest sessions lead to different metabolic responses. This is indicated by the significant improvement from pre to post testing in both sexes and training groups in TTE and TT, significantly (20% group, $p < 0.05$)/ closely significantly (15% group, $p = 0.062$) greater increase in critical power (CP), but only a significant increase and magnitude in change in $VO_2$ peak for males (Figure 4.2). There is the potential that females in Study 3 did not have elevated citrate synthase activity as an adaptation to the training, which may explain why there was no change in $VO_2$ peak post HIT (Vigelso, Andersen, Dela., 2014). Vigelso, Andersen, Dela., (2014) compared HIT and endurance research from 1983-2013 and concluded that increasing citrate synthase activity is linked with an increase in $VO_2$ max and that females do not see the same increase in citrate synthase activity compared to males. This observation may not be reliable, as Vigelso, Andersen, Dela., (2014) point out that there is a lack of female participants in HIT and endurance research from 1983-2013. Kavaliauskas, Aspse, Babraj., (2015) speculated that increasing aerobic demand during HIT could be a key factor for improving endurance performance. Table 4.9 in Study 3 further indicates this to be true as increased total $VO_2$ and $VCO_2$ during rest periods ($VO_2$ $r = 0.47$; $VCO_2$ $r = 0.47$) and

sprints ($VO_2$ r = 0.4) is significantly correlated to increasing $VO_2$ peak in both sexes. $VO_2$ and $VCO_2$ measures may be significantly greater in males than in females (Studies 1, 3), however, males do not increase their $VO_2$ and $VCO_2$ measures greater than females. Table 2.2 in Study 1 indicates that the significantly greater $VO_2$ and $VCO_2$ measures in males is due to a reflection of a greater $VO_2$ peak. This indicates that males and females are working to a similar percentage of their $VO_2$ peak within Study 3. Similar heart rate is consistently recorded between males and females in Study 1 and 3, further indicating that both sexes are exercising at a similar capacity. It is also possible that a greater aerobic demand could be achieved by reducing SR rest by > 20%, which in turn would create a smaller work to rest ratio (Kavaliauskas, Aspse, Babraj., 2015). Therefore, the greater aerobic demand during HIT to create a greater improvement in $VO_2$ peak may only work for males. The highest female percentage of $VO_2$ peak data during SR rest periods in Study 1 was recorded at ~61%, possibly indicating that females have to create an aerobic demand beyond ~61% of their $VO_2$ peak (Table 2.2) in order to increase $VO_2$ peak following HIT with reduced SR rest. There is also the possibility that females in Study 3 and the SR group in Study 2 did not increase their $VO_2$ peak due to the duration of the study (Astorino & Schubert., 2014). Astorino & Schubert., (2014) found that the number of participants that increased their $VO_2$ max following 12 weeks of HIT was ~78%, whereas the number of participants that increased their $VO_2$ max following two weeks of HIT was ~65%. Potentially explaining the significant increases in $VO_2$ peak (~12.1% and ~12.8%) with Yamagishi & Babraj., (2017) following nine weeks of HIT when using male and female participants. Therefore, it is possible that females in Study 3 and the SR group in Study 2 could have increased their $VO_2$ peak if the duration of the study was longer (Astorino & Schubert., 2014).

## 5.3 Considerations of procedure protocols

### 5.3.1 Sex differences in selected resistance

Traditionally within HIT research a resistance of 7.5% BM is used for both males and females (Tables 1.1 and 1.2), with recent HIT research adopting less

resistance (6.5-7%) for female participants (Kavaliauskas, Steer, Babraj., 2016; Yamagishi & Babraj., 2017). The reduced resistance for females might be more appropriate due to the morphological differences between sexes, with females having a greater fat mass and males having a greater muscle mass, which is in keeping with greater PPO (Perez Gomez et al., 2008). A significant interaction (sex*trial) in MPO data from Study 1 (Table 2.1) indicates that females have a greater percentage change from the CS MPO when compared to average trial MPO when compared to males. Although, the post hoc was unable to identify were the significance lies it would suggest it lies within trial 6 (15% reduced SR rest), which shows a ~9.8% decrease in MPO from the CS in females and ~1.2% decrease in males. It would suggest that in Study 1 females should have adopted a lighter resistance to allow them to maintain their CS MPO within trials 1-6 (Billaut & Bishop., 2009). However, in Study 3 the first CS was significantly greater than trials 1-4 average MPO in both sexes and the second CS was not significantly different from sessions 1-8 in both sexes. This could suggest that the greater inability for females to maintain their CS MPO (Study 1) is a reflection of pacing tactics rather than the selected resistance (Wittekind, Micklewright, Beneke., 2011). Given that males and females trial MPO has similar changes from CS one (Table 4.1) in Study 3. However, reducing the resistance for females may aid increase the intensity of the sprints during HIT, which may increase both mitochondrial respiration and content (Granata et al., 2016). By decreasing the intensity (using 7.5% BM resistance), it would suggest that females only increase mitochondrial respiration (Granata et al., 2016). This may explain why females improved in every performance measure apart from $VO_2$ peak in Study 3. This is to be debated as Kavaliauskas, Steer, Babraj., (2016) used a 7% BM resistance during HIT for female participants and saw no change in $VO_2$ peak. However, this could indicate that 7% BM resistance is still too great and needs to be further reduced. Possibly explaining why Yamagishi & Babraj., (2017) found an increase in $VO_2$ peak in both training groups that included males and females, when using 6.5% BM resistance for females and 7.5% BM resistance for males. Comparing Kavaliauskas, Steer, Babraj., (2016) and Yamagishi & Babraj., (2017) research could be unfair as the former study was only two weeks long and the latter study was nine weeks long, which may

affect how many participants respond to increasing their $VO_2$ peak (Astorino & Schubert., 2014).

### 5.3.2 Reliable measure for maintenance of power

In previous research achieving a coefficient of variation (CV) of ≤ 5.2% was used to identify if participants were successful in maintaining sprint speed or MPO (Gliaster et al., 2010; Phillips, Thompson, Oliver., 2014). However, this CV of ≤ 5.2% is based off the MPO for the final three sprints of 10 x 7 sec sprints (0.5 kp resistance separated by 30 sec) and not over all ten sprints (Capriotti, Sherman, Lamb., 1999). Using small data samples of three sprints for CV would create a greater bias and smaller degrees of freedom (Hopkins., 2000), which would suggest that the CV of ≤ 5.2% is an unreliable method for identifying maintenance of MPO. Using CV to calculate the maintenance of MPO also appears to be an unreliable measure for identifying a participant's true reflection of maintaining their MPO. This is suggested by the CS MPO data compared to the decrease in trial average MPO data in Studies 1-3 (Tables 2.1, 3.1, and 4.1), despite CV being ≤ 5.2%. The best example of this is in Study 1 with female trial 6 MPO been ~9.8% lower than the CS MPO but CV is reported as ~3.4%. Therefore, using percentage change from the CS might be a more reliable method for measuring maintenance of MPO or speed. Until further research is conducted regarding what percentage of the CS is classified as reliably maintaining MPO, the present study can only recommend a percentage reduction based on the 3 current studies. In Study 1 the average percentage drop from CS MPO was 3.0%, Study 2 had a percentage drop of 3.3% and Study 3 had a percentage drop of 6.6%. Given that people have a tendency to pace repeated sprints, it may be that the actual value should be lower than the average percentage drop. In addition, given that the standard deviation across the 3 studies for percentage drop is 8% then a decrease less than 2.75% from CS MPO may be appropriate for determining successful self-regulation.

## 5.4 Future direction

### 5.4.1 Maintaining criterion sprint mean power output using self-regulated rest

The above has suggested that participants can maintain a MPO over 10 x 6 sec sprints with an average change of -2.75% from the CS, with Study 1 finding that male participants being able to produce an average MPO that is 2.5% greater than their CS (trial 3; Table 2.1). Given that maintaining power output is speculated as a key factor for seeking to improve endurance and power output testing (Hazell et al., 2010; Lloyd Jones, Morris, Jakeman., 2017), seeking to identify at what percentage participants can maintain of their CS using SR recovery would allow a bench mark for participants to aim towards during their training. At the moment limited research can only indicate that maintaining repeat sprint ability MPO or sprint speed time with a CV of ≤ 5.2% is classified as successfully maintaining maximal MPO or sprint time (Glaister et al., 2010; Phillips, Thompson, Oliver., 2014). Whereas Studies 1-3 found that using CV to identify if a participant is maintaining their MPO is not a true indication of a participant maintaining their true maximal MPO, but rather a reduction from their CS potentially due to pacing tactics. Recruiting a large population (100-300) of male and female participants completing three trials (two familiarisation trials) of 10 x 6 sec sprints (against 7.5% body mass) with the aim of self-regulating their recovery to repeatedly reproduce their CS MPO. Given that participants are likely to pace their efforts during repeat sprint activity (Phillips, Thompson, Oliver., 2014; Study 1-3) and that removing an over-estimation in recovery leads to greater improvements in endurance measures (Studies 2 and 3), then seeking to identify recovery patterns may aid in removing over-estimation of SR recovery. Therefore, within the proposed trial 3 measuring $VO_2$, $VCO_2$, HR, breathing rate, and core body temperature (physiological measures) between each sprint could identify recovery patterns. For example if the above measures reach a plateau during SR recovery this could indicate that the participant is ready to start their next sprint and therefore aid in removing any over-estimation in required recovery. Given that not every participant can maintain their CS sprint MPO, using multiple participants will allow average physiological measures readings for those that can maintain above their CS, with the 2.75%

bracket and below. This will identify 1) if a majority of participants can maintain their CS MPO; and 2) if the physiological measures are affected by participants who can and cannot maintain their CS MPO. In addition to this, performing maximal voluntary contractions prior to the start of trial 3 and post-trial 3 (use an average SR recovery of trial 3 post sprint 10) in the form of a single isometric leg extension whilst using electromyography (EMG) measures. Using EMG measures of root mean squared and medium frequency from the quadriceps pre and post-trial 3 will identify changes in voluntary maximal isometric contraction. If there is no change in EMG measures pre to post and if CS MPO is maintained (≤ 2.75%) then it could suggest that the participants are pacing their efforts, if there is a drop in EMG measures in opposite circumstances then it could indicate that they are not pacing as much. If average MPO is less than CS (≥ 2.75%) and EMG is unchanged then it also suggests pacing. If average MPO is less than CS (≥ 2.75%) and EMG measures are reduced then it could suggest that the participant is unable to maintain their MPO. This study will identify what percentage change from the CS within trial average MPO is classified as maintaining MPO reliably.

### 5.4.2 Effects of resistance during cycle sprints on endurance measures over 12 weeks in females

Study 3 found that females increased their performance in all endurance testing measures but were unable to see an increase in $VO_2$ peak following SR reduced rest (RR (15-20%)) HIT. It is speculated from this thesis and others (Vigelso, Andersen, Dela., 2014) that $VO_2$ peak did not increase due to the potential that females do not see a meaningful increase in citrate synthase activity post HIT, the resistance of the HIT is too great, and the duration of Study 3 is too short. Purely recruiting female participants for this further research study may remove the hypothesis that females do not see as great an increase in citrate synthase activity than males, which is based off a lack of female participants within HIT research (Vigelso, Andersen, Dela., 2014). Testing measures would involve a similar process to Burgomaster et al., (2005), with a testing weeks (pre testing and weeks 4, 8, 12) consisting of $VO_2$ peak, TTE and CP testing, along with muscle biopsy testing. Muscle biopsy testing

would seek to measure resting glycogen (mmol/kg$^{-1}$ dry wt), ATP, PCr and creatine stores (mmol/kg dry wt), along with citrate synthase activity (mol.kg protein$^{-1}$-h$^{-1}$ww). The use of muscle biopsy testing would further explain potential changes in $VO_2$ peak, TTE and CP testing, and disprove the potential hypothesis that females do not see as strong a change in citrate synthase activity and $VO_2$ peak post HIT (Vigelso, Andersen, Dela., 2014). In addition to this, using a long term study (12 weeks) would also identify if increasing the intensity of the sprints during HIT, by using three training groups using 6.5, 7 and 7.5% BM resistance, leads to an increase in citrate synthase activity and $VO_2$ peak over three post testing points (weeks 4, 8 and 12). The HIT within this new research plan would be separated by 15-20% SR RR to identify why females did not experience an increase in $VO_2$ peak in Study 3. This would allow further direction to HIT research and or practitioners to reliably increase $VO_2$ peak in females for performance or health benefits.

### 5.4.3 Human muscle metabolism during self-regulated repeat sprint activity

Hazell et al., (2010) speculates that maintaining power output during HIT might be responsible for similar training adaptations to seeking to decrement power output during HIT and therefore create a larger aerobic demand during the sprint (Bogdanis et al., 1996; Gaitanos et al., 1993). Both Bogdanis et al., (1996) and Gaitanos et al., (1993) have identified what occurs in muscle metabolism and what causes the majority of ATP turn over during 2 x 30 sec (4 min rest) and 10 x 6 sec (30 sec rest) sprints respectively. Despite the differences in sprint durations between the two studies, both these studies found an increased contribution in aerobic metabolism from the first to the final sprint (29-43% Bogdanis et al., 1996), with a significant decrease in PPO (33.4%) and MPO (26.6% (Gaitanos et al., 1993)). However, research is limited on the effects of muscle metabolism when participants are maintaining their power output. Significant increases in $VO_2$, $VCO_2$ and HR from sprint 1/ rest 1 to sprint 10/ rest 9 in Studies 1-3 in both males and females in SR and RR trials when maintaining MPO would suggest that muscle metabolism could be similar to previous research (Bogdanis et al., 1996; Gaitanos et al., 1993). Future

research could complete a similar protocol to Gaitanos et al., (1993) and have participants complete three trials of 10 x 6 sec sprints (7.5% body mass resistance), using SR recovery and aim to maintain MPO. The first two trials would be completed as a familiarisation procedure with trial three involving muscle biopsies. To be consistent with Gaitanos et al., (1993), muscle biopsies would be taken before sprint one, post sprint 1, pre sprint 10 and post sprint 10. With blood samples been taken before sprint one, post sprint 1, post sprint 5, post sprint 10, and 1, 3, 5, 10 minutes after the completion of sprint 10. At these time points the muscle biopsies and blood samples will measure changes in blood lactate, blood pH. ATP, adenosine diphosphate, adenosine monophosphate, PCr, creatine, glycogen, and glucose 6 phosphate. These muscle biopsies would allow an estimate of ATP production from PCr and ATP, glycolysis, and aerobic metabolism between sprint 1 and sprint 10. If maintaining power output and decreasing power output (due to an insufficient rest) leads to similar muscle metabolism then it would indicate maintaining power output during HIT is responsible for increases in performance measures, as speculated by Hazell et al., (2010). This would also indicate that the use of specific work to rest ratios during HIT are not vital and or could create specific work to rest ratios for each individual based off their current level of fitness.

### 5.4.4 Is self-regulated rest controlled by peripheral or central feedback?

There appears to be little research specifically based on how self-selected rest periods during exercise is regulated. Within this thesis it is hypothesised that SR rest, when a participant has an understanding of been able to reproduce their maximal MPO, is mainly controlled by afferent feedback with some contribution from central feedback possibly in the latter sprints/ recoveries (Kent-Braun., 1999). Studies 1-3 found a consistent significant increase in $VO_2$, $VCO_2$ and HR from the early stages to the latter stages of a SR trial in Studies 1-3, and the greater increase in SR recovery from rest 1 to rest 9 (significantly greater in trial 3 Study 3). This could suggest that SR recovery is controlled by afferent feedback (Gallagher et al., 2001) but does not take into account any possible increase in central feedback. Identifying how SR recovery is controlled could aid the understanding of when a participant would be ready to reproduce their

maximal effort and potentially remove any over-estimation in rest that may occur (Phillips, Thompson, Oliver., 2014; Study 1). Removing this over-estimation in SR recovery duration might be responsible for greater increases in endurance measures in Study 3 compared to Study 2. Previous research has found that during moderate cycling (7 min at 20% $VO_2$ max) found significantly greater measures of lactate and noradrenaline concentration, HR, and perceived exertion when participants neuromuscular pathways had been blocked when compared to the control period (Gallagher et al., 2001). Suggesting that perceived exertion is regulated by afferent feedback, given that increases in HR and increased glycolysis use is related to greater perceived exertion (Marcora., 2009). Neuromuscular junction had been blocked by participants been administered a small dose of curare, which would not affect afferent pathways (Gallagher et al., 2001). In future research, participants would undergo a trial of maintaining MPO using SR rest under no condition and then under a small dose of curare. Between the two conditions, SR recovery duration between sprints and the MPO of each sprint would be recorded and compared against. If maintaining MPO and SR recovery duration is unaffected under the curare condition compared to the control condition then it would suggest that SR recovery is controlled by afferent feedback. If participants SR recovery is increased and or MPO decreases under the curare condition then it would suggest that SR recovery is controlled by central feedback.

## 5.5 Overall conclusion

The overall aims to this thesis were: 1) To determine if males and females can maintain mean power output during repeated sprints with self-regulated rest (Study one). 2) To identify male and female response in mean power output when self-regulated rest is reduced (Study one). 3) Compare endurance and Wingate power output adaptations to HIT with a fixed rest (30 sec) or self-regulated rest (Study two). 4) To identify if reproducibility of mean power output is correlated to endurance adaptation to HIT (Studies two and three). 5) To compare the magnitude in change of $VO_2$ peak, time to exhaustion, 10km time trial, and critical power between 15 and 20% reduced self-regulated rest during repeat sprint training between males and females (Study three). It was found

that males can maintain their MPO greater than females when using SR rest, and males can also maintain their MPO greater than females when SR rest is reduced by 10 and 15% (Study one). Using SR rest during a HIT intervention leads to greater improvements in TTE, TT and Wingate power output compared to using a fixed rest (1:5 work to rest ratio), whereas a fixed rest leads to greater improvements in $VO_2$ peak (Study two). Reducing SR rest by 15 and 20% in a HIT intervention leads to greater improvements in both sexes for $VO_2$ peak (no change in females), TTE and TT when compared to SR rest during HIT in Study two. CP also increased significantly and near significantly for both 20 and 15% reduced SR rest groups respectively (Study three). Studies two and three were unable to find an association between maintaining MPO and increasing endurance measures or Wingate power output measures. However, Study 3 indicates that increasing $VO_2$ peak is associated with increasing aerobic demand during HIT. Correlation data indicates that TTE and TT improvements are due to improvements in power output and CP (Study two and three).

## 5.6 Overall practical implications

This thesis is able to offer the following practical implications to practitioners: 1) using CV method of ≤ 5.2% to identify if a participant can maintain their MPO when using SR rest is unreliable as it does not take into account the participant's greatest single MPO value. Until further research is developed this thesis can only recommend that maintaining ≤ 2.75% of a participant's CS MPO is deemed as successfully maintaining MPO within a trial or session. 2) Females may require a reduction in the typically prescribed 7.5% of their body mass as a resistance to maintain their MPO when using SR rest, due to morphological differences between males and females (Perez Gomez et al., 2008). 3) Using SR rest can lead to greater adaptations in TTE, TT and Wingate power output testing, and also increase haemoglobin measures greater than using a fixed work:rest ratio (1:5). 4) Practitioners seeking to increase $VO_2$ peak should use a work to rest ratio of 1:3 – 1:8 (Kavaliauskas, Aspe, Babraj., 2015). 5) Reducing SR by 15 and 20% leads to greater adaptations in $VO_2$ peak, TTE and TT compared to non-reduced SR during HIT. However, females see no change in $VO_2$ peak when SR rest is reduced by 15 or 20%. Based off previous

research (Yamagishi & Babraj., 2017), this thesis can only suggest that reducing the cycling resistance to 6.5% of a female participant's body mass may increase $VO_2$ peak. 6) Increasing $VO_2$ peak is significantly correlated to increasing aerobic demand during HIT. Therefore, practitioners should actively seek to achieve a greater aerobic demand during HIT for their athletes if they are seeking to increase their $VO_2$ peak. 7) This thesis provides further evidence that using high intensity sprints as short as 6 sec leads to significant improvements in endurance measures.

# 6 Reference List

Adan. A. (1994). Chronotype and personality factors in the daily consumption of alcohol and psychostimulants. *Addiction*. Vol 89(4) pp: 455-517.

Adamson. J. W., and Finch. C. A. (1975). Hemoglobin function, oxygen affinity, and erythropoietin. *Annual Review of Physiology*. Vol 37 pp: 351-420.

Allemeier. C. A., Fry. A. C., Johnson. P., Hikida. R. S., Hagerman. F. C., and Staron. R. S. (1994). Effects of sprint cycle training on human skeletal muscle. *Journal of Applied Physiology*. Vol 77(5) pp: 2385-2390.

Amann. M. (2011). Central peripheral fatigue: interaction during cycling exercise in humans. *Medicine & Science in Sports & Exercise*. Vol 43(11) pp: 2039-2084.

Amann. M. (2012). Significance of group III and IV muscle afferents for the endurance exercising human. *Clinical and Experimental Pharmacology and Physiology*. Vol 39(9) pp: 831-835.

Amann. M., and Dempsey. J. A. (2008). Locomotor muscle fatigue modifies central motor drive in healthy humans and imposes a limitation to exercise performance. *The Journal of Physiology*. Vol 586(1) pp: 161-234.

Astorino. T. A., and Schubert. M. M. (2014). Individual responses to completion of short-term and chronic interval training: a retrospective study. *PLoS One*. Vol 9(5): e97638.

Ayalon. A., Inbar. O., and Bar-Or. O. (1974). Relationships among measurements of explosive strength and anaerobic power. *International Series on Sport Sciences. Biomechanics IV*. Vol 1 pp: 572-577.

Baden. D., McLean. T., Tucker. R., Noakes. T., St Clair Gibson. A., and Hunter. A. (2005) Effect of anticipation during unknown or unexpected exercise duration on rating of perceived exertion, affect, and physiological function. *British Journal of Sports Medicine*. Vol 39(10) pp: 742-746.

Baechle. T. R., and Earle. R. W. (2008). *Essentials of strength training and conditioning.* Champaign. Human Kinetics.

Bagley. L., Slevin. M., Bradburn. S., Liu. D., Murgatroyd. C., Morrissey. G., Carroll. M., Piasecki. M., Gilmore. W. S., and McPee. J. S. (2016). Sex differences in the effects of 12 weeks sprint interval training on body fat mass and the rates of fatty acid oxidation and VO2max during exercise. *BMJ Open Sport & Exercise Medicine.* Vol 2(1) pp: 1-8.

Bai. Z., Wang. K., and Wong. (2011). The mean-variance ratio test – A complement to the coefficient of variation test and the Sharpe ratio test. *Statistics and Probability Letters.* Vol 81 pp: 1078-1085.

Balsom. P. D., Gaitanos. G. G., Soderlund. K., and Ekblom. B. (1999). High-intensity exercise and muscle glycogen availability in humans. *Acta Physiologica Scandinavica*. Vol 165(4) pp: 337-45.

Balsom. P. D., Sejer. J. Y., Sjodin. B., and Ekblom. B. (1992). Maximal intensity intermittent exercise: effect of recovery duration. *International Jounral of Sports Medicine*. Vol 13 pp: 528-533.

Bassett. D. R., and Howley. E. T. (1997). Maximal oxygen uptake: “classical” versus “contemporary” viewpoints. *Medicine & Science in Sports & Exercise*. Vol 29(5) pp: 591-603.

Bassett. D. R., and Howley. E. T. (2000). Limiting factors for maximum oxygen uptake and determinants of endurance performance. *Medicine and Science in Sports and Exercise*. Vol 32(1) pp: 70-84.

Baumeister. R. F., and Vohs. K. D. (2007). *Handbook of self-regulation: research, theory, and applications*. New York. The Guilford Press.

Bell. K. E., Séguin. C., Parise. G., Baker. S. K., and Phillips. S. M. (2015). Day-to-day changes in muscle protein synthesis in recovery from resistance, aerobic, and high-intensity interval exercise in older men. *The Journals of Gerontology. Series A, Biological Sciences and Medical Sciences.* Vol 70(8) pp: 1024-1029.

Bergstrom. H. C., Housh. T. J., Zuniga. J. M., Camic. C. L., Traylor. D. A., Schmidt. R. J., and Johnson. G. O. (2012). A new single work bout test to

estimate critical power and anaerobic work capacity. *Journal of Strength & Conditioning Research*. Vol 26(3): 656-663.

Billaut. F., and Basset. F. A. (2007). Effect of different recovery patterns on repeated-sprint ability and neuromuscular responses. *Journal of Sports Sciences*. Vol 25(8) pp: 905-913.

Billaut. F., and Bishop. D. J. (2012). Mechanical work accoutns for sex differences in fatigue during repeated sprints. *European Journal of Applied Physiology*. Vol 112(4) pp: 1429-1465.

Billaut. F., and Bishop. D. (2009). Muscle fatigue in males and females during multiple-sprint exercise. *Sports Medicine*. Vol 39(4) pp: 257-278.

Billaut. F., Bishop. D., Schaerz. S. D., and Noakes. T. D. (2011). Influence of knowledge of sprint number on pacing during repeated-sprint exercise. *Medicine & Science in Sports & Exercise*. Vol 43(4) pp: 665-8.

Bishop. D., Edge. J., Thomas. C., and Mercier. J. (2008). Effects of high-intensity training on muscle lactate transporters and post exercise recovery of muscle lactate and hydrogen ions in women. *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology*. Vol 295 pp: 1991-1998.

Bishop. D. J. (2012). Fatigue during intermittent-sprint exercise. *Proceedings of the Australian Physiological Society*. Vol 43 pp: 9-15.

Bogdanis. G. C., Nevill. M. E., Boobis. L. H., and Lakomy. H. K. (1996). Contribution of phosphocreatine and aerobic metabolism to energy supply during repeated sprint exercise. *Journal of Applied Physiology*. Vol 80(3) pp: 876-960.

Bogdanis. G. C., Nevill. M. E., Lakomy. H. K., and Boobis. L. H. (1998). Power output and muscle metabolism during and following recovery from 10 and 20 s of maximal sprint exercise in humans. *Acta Physiological Scandinavian.* Vol 163(3) pp: 261-333.

Brooks. G. A., Fahey. T. D., and Baldwin. K. M. (2004). *Exercise physiology: human bioenergetics and its applications*. New York. The McGraw-Hill Companies Inc.

Bulbulian. R., Wilcox. A. R., and Darbos. B .L. (1986). Anaerobic contribution to distance running performance of trained cross-country athletes. *Medicine & Science in Sports & Exercise*. Vol 18 pp: 107-113.

Burgomaster. K. A., Heigenhauser. J. F., and Gibala. M. J. (2006). Effect of short-term sprint interval training on human skeletal muscle carbohydrate metabolism during exercise and time trial performance. *Journal of Applied Physiology*. Vol 100 pp: 2041-2047.

Burgomaster. K. A., Howarth. K. R., Phillips. S. M., Rakobowchuk. M., MacDonald. M. J., McGee. S. L., and Gibala. M. J. (2008). Similar metabolic adaptations during exercise after low volume sprint interval and traditional endurance training in humans. *Journal of Physiology*. Vol 586(1) pp: 151-160.

Burgomaster. K. A., Hughes. S. C., Heigenhauser. G. J. F., Bradwell. S. N., and Gibala. M. (2005). Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans. *Journal of Applied Physiology*. Vol 98 pp: 1985-1990.

Burnley. M., Doust. J. H., and Vanhatalo. A. (2006). A 3-min all-out test to determine peak oxygen uptake and the maximal steady state. *Medicine and Science in Sports and Exercise*. Vol 38(11) pp: 1995-2003.

Brooks. G. A. (2000). Intra-and extra-cellular lactate shuttles. *Medicine & Science in Sports & Exercise*. Vol 32(4) pp: 790-799.

Camera. D. M., Hawley. J. A., and Coffey. V. G. (2015). Resistance exercise with low glycogen increases p53 phosphorylation and PGC-1alpha mRNA in skeletal muscle. *European Journal of Applied Physiology*. Vol 115(6) pp: 1185-1279.

Capriotti. P. V., Sherman. M. V., and Lamb. D. R. (1999). Reliability of power output during intermittent high-intensity cycling. *Medicine & Science in Sports & Exercise*. Vol 31(6) pp: 913-915.

Christensen. P. M., Jacobs. R. A., Bonne. T., Flück. D., Bangsbo. J., and Lundby. C. (2016). A short period of high-intensity interval training improves skeletal muscle mitochondrial function and pulmonary oxygen uptake kinetics. *Journal of Applied Physiology*. Vol 120 pp: 1319-1327.

Creer. A. R., Ricard. M. D., Conlee. R. K., Hoyt. G. L., and Parcell. A. C. (2004). Neural, metabolic, and performance adaptations to four weeks of high intensity sprint-interval training in trained cyclists. *International Journal of Sports Medicine*. Vol 25(2) pp: 92-98.

Cohen. J. (1992). A power primer. *Psychological Bulletin*. Vol 112(1) pp: 155-164.

Costill. D. L., Fink. W. J., and Pollock. M. L. (1976). Muscle fiber composition and enzyme activities of elite distance runners. *Medicine & Science in Sports & Exercise*. Vol 8(2) pp: 96-100.

Dantas. J. L., Pereira. G., and Nakamura. F. Y. (2015). Five-kilometers time trial: preliminary validation of a short test for cycling performance evaluation. *Asian Journal of Sports Medicine*. Vol 6(3) e23802.

Davis. J. M. (1995). Central and peripheral factors in fatigue. *Journal of Sports Sciences*. Vol 13(1) pp: 49-53.

Dawson. B., Goodman. C., Lawrence. S., Preen. D., Polglaze. T., Fitzsimons. M., and Fournier. P. *Scandinavian Journal of Medicine & Science in Sports*. Vol 7(4) pp: 206-219.

Decorte. N., Lafaix. P. A., Millet. G. Y., Wuyam. B., and Verges. S. (2012). Central and peripheral fatigue kinetics during exhaustive constant-load cycling. *Scandinavian Journal of Medicine & Science in Sports*. Vol 22 (3) pp: 381-472.

Driller. M. W. (2012). The reliability of a 30-minute performance test on a Lode cycle ergometer. *Journal of Science and Cycling*. Vol 1(2) pp: 21-27.

Edwards. A. M., and Polman. R. C. (2013). Pacing and awareness: brain regulation of physical activity. *Sports Medicine*. Vol 43(1) pp: 1057-1065.

Esbjornsson-Lijedahl. M., Bodin. K., and Jansson. E. (2002). Smaller ATP reduction in women than in men by repeated bouts of sprint exercise. *Journal of Applied Physiology*. Vol 93(3) pp: 1075-1083.

Esbjörnsson-Lijedahl. M., Hellsten-Westling. Y., Balsom. P. D., Sjödin. B., and Jansson. E. (1993). Muscle fibre type changes with sprint training: effect of training pattern. *Acta Physiologica Scandinavica*. Vol 149(2) pp: 245-251.

Fitzsimmons. M., Dawson. B., Ware. D., and Wilkinson. A. (1993). Cycling and running tests of repeated sprint ability. *Australian Journal of Science and Medicine in Sport*. Vol 25 pp: 82-87.

Forbes. S. C., Slade. J. M., and Meyer. R. A. (2008). Short-term high-intensity interval training improves phosphocreatine recovery kinetics following moderate-intensity exercise in humans. *Applied Physiology, Nutrition and Metabolism*. Vol 33 pp: 1124-1131.

Francis. J. T., Quinn. T. J., Amann. M., and LaRoche. D. P. (2010). Defining intensity domains from the end power of a 3-min all-out cycling test. *Medicine and Science in Sports and Exercise*. Vol 42(9) pp: 1769-1844.

Froyd. C., Beltrami. F. G., Millet. G. Y., and Noakes. T. D. (2016) Central regulation and neuromuscular fatigue during exercise of different durations. *Medicine & Science in Sports & Exercise*. Vol 48(6) pp: 1042-1074.

Froyd. C., Millet. G. Y., and Noakes. T. D. (2013). The development of peripheral fatigue and short-term recovery during self-paced high-intensity exercise. *Journal of Physiology*. Vol 59(5) pp: 1399-1385.

Fulco. C. S., Rock. P. B., Muza. S. R., Lammi. E., Cymerman. A., Butterfield. G., Moore. L. G., Braun. B., and Lewis. S. F. (1999). Slower fatigue and faster recovery of the adductor pollicis muscle in women matched for strength with men. *Acta Physiologica Scandinavian*. Vol 167 pp: 233-239.

Gallagher. K. M., Fadel. P. J., M. Strømstad. M., Ide. K., Smith. S. A., Querry. R. G., Raven. P. B., and Secher. N. H. (2001). Effects of partial neuromuscular blockade on carotid baroreflex function during exercise in humans. *The Journal of Physiology*. Vol 533(3) pp: 861-870.

Gandevia. S. C. (2001). Spinal and supraspinal factors in human muscle fatigue. *Physiological Reviews*. Vol 81(4) pp: 1725-1814.

Gaitanos. G. C., Williams. C., Boobis. L. H., and Brooks. S. (1993). Human muscle metabolism during intermittent maximal exercise. *Journal of Applied Physiology*. Vol 75: pp: 712-719.

Gladden. B. L. (2000). Muscle as a consumer of lactate. *Medicine & Science in Sports & Exercise*. Vol 32(4) pp: 764-771.

Glenmark. B., Hedberg. G., and Jansson. E. (1992). Change in muscle fibre type from adolescence to adulthood in women and men. *Acta Physiologica Scandinavica*. Vol 146(2) pp: 251-259.

Gibala. M. J., Little. J. P., van Essen. M., Wilkin. G. P., Burgomaster. K. A., Safdar. A., Raha. S., and Tarnopolsky. M. A. (2006). Short-term sprint interval versus traditional endurance training: similar initial adaptations in human skeletal muscle and exercise performance. *Journal of Physiology*. Vol 575(3) pp: 901-911.

Glaister. M. (2005). Multiple sprint work. Physiological responses, mechanisms of fatigue and the influence of aerobic fitness. *Sports Medicine*. Vol 35(9) pp: 757-777.

Glaister. M., Witmer. C., Clarke. D. W., Guers. J. J., Heller. J. L., and Moir. G. L. (2010). Familiarization, reliability, and evaluation of a multiple sprint running test using self-selected recovery periods. *Journal of Strength and Conditioning Research*. Vol 24 pp: 3296-3301.

Goodall. S., Charlton. K., Howatson. G., and Thomas. K. (2015). Neuromuscular fatigability during repeated-sprint exercise in male athletes. *Medicine & Science in Sports & Exercise*. Vol 47(3) pp: 528-564.

Gosselin. L. E., Kozlowski. K. F., DeVinney-Boymel. L., and Hambridge. C. (2012). Metabolic response of different high-intensity aerobic interval exercise protocols. *Journal of Strength and Conditioning Research*. Vol 26 pp: 2866-2871.

Granata. C., Oliveira. R. S., Little. J. P., Renner. K., and Bishop. D. J. (2016). Training intensity modulates changes in PGC-1a and p 53 protein content and mitochondrial respiration, but not markers of mitochondrial content in human skeletal muscle. *The FASEB Journal*. Vol 30 pp: 959-970.

Green. H., Grant. S., Bombardier. E., and Ranney. D. Initial aerobic power does not alter muscle metabolic adaptations to short-term training. *American Journal of Physiology*. Vol 277(1) pp: 39-48.

Gurd. B. J., Giles. M. D., Bonafiqlia. J. T., Raleigh. J. P., Boyd. J. C., Ma. J. K., Zelt. J. G., and Scribbans. T. D. (2016). Incidence of nonresponse and individual patterns of response following sprint interval training. *Applied Physiology, Nutrition, and Metabolism*. Vol 41(3) pp: 229-263.

Hargreaves. M., and Spriet. L. L. (2006). *Exercise Metabolism*. Second addition. Human Kinetics.

Harris. R. C., Edward. R. H., Hultman. E., Nordesjo. L. O., Nylind. B., and Sahlin. K. (1976). The time course of phosphorylcreatine resynthesis during recovery of the quadriceps muscle in man. *European Journal of Physiology*. Vol 367 pp: 137–142.

Hasimoto, T., Hussien, R., Brooks. G. A. (2006). Colocalization of $MTC_1$, CD147, and LDH in mitochondrial inner membrane of L6 muscle cells: evidence of mitochondrial lactate oxidation complex. *American Journal of Physiology*. Vol 290 pp: 1237-1244.

Haskell. W. L., Lee. I., Pate. R. R., Powell. K. E., Blair. S. N., Franklin. B. A., Macera. C. A., Heath. G. W., Thompson. P. D., and Bauman. A. (2007). Physical activity and public health: updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Physical Activity and Public Health*. Vol 116 pp: 1081-1093.

Haverty. M., Kenny. W. L., and Hodgson. J. L. (1988). Lactate and gas exhcnaces responses to incremental and steady state running. *British Journal of Sports Medicine*. Vol 22(2) pp: 51-54.

Hazell. T. J., MacPherson. R. E. K., Gravelle. B. M. R., and Lemon. P. W. R. (2010). 10 or 30-s sprint interval training bouts enhance both aerobic and anaerobic performance. *European Journal of Applied Physiology*. Vol 110(1) pp: 153-160.

Hicks. A. L., Kent-Braun. J., and Ditor. D. S. (2001). Sex differences in human skeletal muscle fatigue. *Exercise & Sport Sciences Reviews*. Vol 29(3) pp: 109-112.

Holloszy. J. O., and Coyle. E. F. (1984). Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *Journal of Applied Physiology*. Vol 56 pp: 831-838.

Hopkins. W. G. (2000). Measures of reliability in sports medicine and science. *Sports Medicine*. Vol 30(1) pp: 1-15.

Hopkins. W. G., Schabort. E. J., and Hawley. J. A. (2001). Reliability of Power in Physical Performance Tests. *Sports Medicine*. Vol 31(3) pp: 211-245.

Hughes. D. C., Ellefsen. S., and Baar. K. (2017). Adaptations to endurance and strength training. *Cold Spring Harbor Perspectives in Medicine*. a029769. doi: 10.1101/cshperspect.a029769.

Hultman. E., Bergstrom. J., and Anderson. N. M. (1967). Breakdown and resynthesis of phosphorylcreatine and adenosine triphosphate in connection with muscular work in man. *Scandinavian Journal of Clinical & Laboratory Investigation*. Vol 19 pp: 56–66.

Hultman., E and Sjöholm. H. (1983). Energy metabolism and contraction force of human skeletal muscle in situ during electrical stimulation. *Journal of Physiology*. Vol 345 pp: 525-557.

Hunter. S. K. (2014). Sex differences in human fatigability: mechanisms and insight to physiological responses. *Acta Physiologica*. Vol 210(4) pp: 768-22.

Hureau. T. J., Olivier. N., Millet. G. Y., Meste. O., and Blain. G. M. (2014). Exercise performance is regulated during repeated sprints to limit the

development of peripheral fatigue beyond a critical threshold. *Experimental Physiology*. Vol 99(7) pp: 951-963.

Jacobs. I., Esbjörnsson. M., Sylvén. C., Holm. I., and Jansson. E. (1987). Sprint training effects on muscle myoglobin, enzymes, fiber types, and blood lactate. *Medicine & Science in Sports & Exercise*. Vol 19(4) pp: 368-442.

Jakeman. J., Adamson. S., and Babraj. J. (2012). Extremely short duration high-intensity training substantially improves endurance performance in triathletes. *Applied Physiology, Nutrition and Metabolism*. Vol 37 pp: 976-981.

Jansson. E., Esbjörnsson. M., Holm. I., and Jacobs. I. (1990). Increase in the proportion of fast-twitch muscle fibres by sprint training in males. *Acta Physiologica Scandinavica*. Vol 140(3) pp: 359-422.

Jeukendrug. A., Saris. W. H., Brouns. F., and Kester. A. D. (1996). A new validated endurance performance test. *Medicine and Science in Sports and Exercise*. Vol 28(2) pp: 266-336.

Joyner. M .J., and Coyle. E. F. (2008). Endurance  exercise performance: the during high-intensity cycling in warm, humid conditions. *European Journal of Applied Physiology*. Vol 84 pp:115-121.

Juel. C., and Halestrap. A. P. (1999). Lactate transport in skeletal muscle – role and regulation of monocarboxylate transporter. *Journal of Physiology*. Vol 517(Pt3) pp: 633-642.

Juel. C., Klarskov. C., Nielsen. J. J., Krushtrup. P., Mohr. M., and Bangsbo. J. (2003). Effect of high-intensity intermittent training on lactate and $H^+$ release from human skeletal muscle. *American Journal of Physiology*. Vol 286(2) pp: 245-251.

Jones. A. M., and Burnley. M. (2009). Oxygen uptake kinetics: an underappreciated determinant of exercise performance. *Human Kinetics Journals*. Vol 4(4) pp: 524-532.

Jones. A. M., Vanhatalo. A., Burnley. M., Morton. R. H., and Poole. D. C. (2010). Critical power: implications for determination of $VO_2$ max and exercise tolerance. *Medicine & Science in Sports & Exercise*. Vol 42(10) pp: 1876-1890.

Jones. N. L., McCartney. N., Graham. T., Spriet. L. L., Kowalchuk. J. M., Heigenhauser. G. J., Sutton. J. R. (1985). Muscle performance and metabolism in maximal isokinetic cycling at slow and fast speeds. *Journal of Applied Physiology*. Vol 59(1) pp: 132-138.

Kanstrup. I., and Ekblom. B. (1984). Blood volume and hemoglobin concentration as determinants of maximal aerobic power. *Medicine and Science in Sports and Exercise*. Vol 16(3) pp: 256-262.

Kavaliauskas. M., Aspe. R. R., and Babraj. J. (2015). High-intensity cycling training: the effect of work-to-rest intervals on running performance measures. *Journal of Strength & Conditioning Research*. Vol 29(8) pp: 2229-2237.

Kavaliauskas. M., Steer. T. P., and Babraj. J. A. (2016). Cardiorespiratory fitness and aerobic performance adaptations to a 4-week sprint interval training in young healthy untrained females. *Sports Sciences for Health*. Vol 13(1) pp: 17-23.

Kay. D., Marino. F. E., Cannon. J., St Clair Gibson. A., Lambert. M. I., and Noakes. T. D. (2001). Evidence for neuromuscular fatigue during high-intensity cycling in warm humid conditions. *European Journal of Applied Physiology*. Vol 84 pp: 115-121.

Kent-Braun. J. A., and Alexander. V. N. G. (2000). Skeletal muscle oxidative capacity in young and older women and men. *Journal of Applied Physiology*. Vol 89 pp: 1072-1078.

Kent-Braun. J. A. (1999). Central and peripheral contributions to muscle fatigue in humans during sustained maximal effort. (1999). *European Journal of Applied Physiology and Occupational Physiology*. Vol 80(1) pp: 57-63.

Knechtel. B., Muller. G., Willmann. F., Kotteck. K., Eser. P., and Knecht. H. (2004). Fat oxidation in men and women endurance athletes in running and cycling. *International Journal of Sports Medicine*. Vol 25 pp: 38-44.

Knuiman. P., Hopman. M. T. E., and Mensink. M. (2015). Glycogen availability and skeletal muscle adaptations with endurance and resistance exercise. *Nutrition & Metabolism*. Vol 12(59) pp 1-12.

Kraemer. W. J., Fleck. S. J., and Evans. W. J. (1996). Strength and power training: physiological mechanisms of adaption. *Exercise and Sport Sciences Reviews*. Vol 24 pp: 363-460.

Laurent. C. M., Green. J. M., Bishop. P. A., Sjokvist. J., Schumacker. R. E., Richardson. M. T., and Curtner-Smith. M. (2010). Effect of gender on fatigue and recovery following maximal intensity repeated sprint performance. *Journal of Sports Medicine, Physiology and Fitness*. Vol 50(3) pp: 243-296.

Laursen. P. B., Francis. G. T., Abbiss. C. R., Newton. M. J., and Nosaka. K. (2007). Reliability of time to exhaustion versus time trial running tests in runners. *Medicine & Science in Sports & Exercise.* Vol 39(8) pp: 1374-1383.

Laursen. P. B., and Jenkins. D. G. (2002). The scientific basis for high-intensity interval training. Optimising training programmes and maximising performance in highly trained endurance athletes. *Sports Medicine Australia*. Vol 32(1) pp: 53-73.

Linossier. M. T., Denis. C., Dormois. D., Geyssant. A., and Lacour. J. R. (1993). Ergometric and metabolic adaption to a 5-s sprint training programme. *European Journal of Applied Physiology*. Vol 67 pp: 408-414.

Linossier. M. T., Dormois. D., Perier. C., Frey. J., Geyssant. A., and Denis. C. (1997). Enzyme adaptations of human skeletal muscle during bicycle short-sprint training and detraining. *Acta Physiologica*. Vol 161(4) pp: 439-445.

Little. P. J., Safdar. A., Wilkin. G. P., Tarnopolsky. M. A., and Gibala. M. J. (2010). A practical model of low-volume high-intensity interval training induces mitochondrial biogenesis in human skeletal muscle: potential mechanisms. *The Journal of Physiology*. Vol 588(6) pp: 1011-1022.

Lloyd Jones. M. C., Morris. M. G., and Jakeman. J. R. (2017). Impact of time and work:rest ratio matched sprint interval training programmes on

performance: A randomised controlled trial. *Journal of Science and Medicine in Sport*. Vol 17 pp: 30361-30365.

Loftin. M., Sothern. M., Warren. B., and Udall. J. (2004). Comparison of $VO_2$ peak during treadmill and cycle ergometry in severely overweight youth. *Journal of Sports Science and Medicine*. Vol 3(4) pp: 554-560.

MacDougall. J. D., Hicks. A. L., MacDonald. J. R., McKelvie. R. S., Green. H. J., and Smith. K. M. Muscle performance and enzymatic adaptations to sprint interval training. *Journal of Applied Physiology*. Vol 84(6) pp: 2138-2180.

MacLaren. D., and Morton. J. (2012). *Biochemistry for sport and exercise metabolism*. Wiley-Blackwell.

Magill. R. A. (2010). *Motor learning and control: concepts and applications*. New York. The McGraw-Hill Companies.

Mairbaurl. H. (2013). Red blood cells in sports: effects of exercise and training on oxygen supply by red blood cells. *Frontiers in Physiology*. Vol 4 pp: 1-13.

Marcora. S. (2009). Perception of effort during exercise is independent of afferent feedback from skeletal muscles, heart, and lungs. *Journal of Apllied Physiology*. Vol 106 pp: 2060-2062.

Marsh. A. P., Martin. P. E. (1997). Effects of cycling experience, aerobic power, and power output on preferred and most economical cycling cadences. *Medicine & Science in Sports & Exercise*. Vol 29(9) pp: 1225-1232.

Maughan. R., and Gleeson. M. (2004). The biochemical basis of sports performance. New York, Oxford University Press Inc.

McCartney. N., Spriet. L. L., Heigenhauswer. G . J., Kowalchuk. J. M., Sutton. J. R., and Jones. N. L. (1986). Muscle power and metabolism in maximal intermittent exercise. *Journal of Applied Physiology*. Vol 60 pp: 1164-1169.

Meeusen. R., Watson. P., Hasegawa. H., Roelands. B., and Piacentini. M. F. (2006). *Sports Medicine*. Vol 36(10) pp: 881-909.

Mendez-Villanueva. A., Edge. J., Suriano. R., Hamer. P., and Bishop. D. (2012). The recovery of repeated-sprint exercise is associated with PCr resynthesis, while muscle pH and EMG amplitude remain depressed. *PLoS One*. Vol 7(12): e51977.

Mendez-Villanueva. A., Hamer. P., and Bishop. (2008). Fatigue in repeated-sprint exercise is related to muscle power factors and reduced neuromuscular activity. *European Journal of Applied Physiology*. Vol 103 pp: 411-419.

Menz. V., Strobl. J., Faulhaber. M., Gatterer. H., and Burtscher. M. (2015). Effect of 3-week high-intensity interval training on $VO_{2max}$, total haemoglobin mass, plasma and blood volume in well-trained athletes. *European Journal of Applied Physiology*. Vol 115 pp: 2349-2356.

Morris. S. S., Ruel. M. T., Cohen. R. J., Dewey. K. G., Brière. B., and Hassan. M. N. (1999). Precision, accuracy, and reliability of hemoglobin assessment with use of capillary blood 1-4. *The American Journal of Clinical Nutrition*. Vol 69(6) pp: 1243-1248.

Nevill. M. E., Boobis. L. H., Brooks. S., and Williams. C. (1989). Effect of training on muscle metabolism during treadmill sprinting. *Journal of Applied Physiology*. Vol 67(6) pp: 2376-2382.

Noakes. T. D. (1988). Implications of exercise testing for prediction of athletic performance: A contemporary perspective. *Medicine & Science in Sports & Exercise*. Vol 20 pp: 319-330.

Noakes. T. D., Peltonen. J. E., and Rusko. H. K. (2001). Evidence that a central governor regulates exercise performance during acute hypoxia and hyperoxia. *The Journal of Experimental Biology*. Vol 204(18) pp: 3225-3234.

Ørtenblad. N., Lunde. P. K., Levin. K., Andersen. J. L., and Pedersen. P. K. (2000). Enhanced sarcoplasmic reticulum $Ca^{2+}$ release following intermittent sprint training. *American Journal of Physiology. Regulatory, integrative and comparative physiology*. Vol 279 pp: 152-160.

Ørtenblad. N., Nielsen. J., Saltin. B., and Holmberg. H. (2011). Role of glycogen availability sarcoplasmic reticulum $Ca^{2+}$ kinetics in human skeletal muscle. *Journal of Physiology*. Vol 589(3) pp: 711-725.

Paavolainen. L., Häkkinen. K., Hämäläinen. I., Nummela. A., and Rusko. H. (1999). Explosive-strength training improves 5-km running time by improving running economy and muscle power. *Journal of Applied Physiology*. Vol 86(5) pp: 1527-1560.

Parolin. M. L., Chesley. A., Matsos. M. P., Spriet. L. L., Jones. N. L., and Heigenhauser. G. J. F. (1999). Regulation of skeletal muscle glycogen phosphorylase and PDH during maximal intermittent exercise. *The American Journal of Physiology*. Vol 277 pp: 890-900.

Patton. J. F., Murphy. M. M., and Frederick. F. A. (1985). Maximal power outputs during the Wingate anaerobic test. *International Journal of Sports Medicine*. Vol 6(2) pp: 82-87.

Pereira. G., Martins de Souza. D., Reichert. F. F., and Smirmaul. B. P. C. (2014). Evolution of perceived exertion concepts and mechanisms: a literature review. *Revista Brasileira de Cineantropometria e Desempenho Humano.* Vol 16(15) pp: 579-587.

Perez-Gomez, J, Rodriguez, GV, Ara, I, Olmedillas, H, Chavarren, J, Gonzalez-Henriquez, JJ, Dorado, C, and Calbet, JAL (2008). Role of muscle mass on sprint performance: gender differences? *Eur J Appl Physiol* 102: 685-694, 2008.

Periasamy. M., and Kalyanasundaram. A. (2007). SERCA pump isoforms: their role in calcium transport and disease. *Muscle and Nerve*. Vol 35 pp: 430-442.

Perry. C. G. R., Heigenhauser. G. J. F., Bonen. A., and Spriet. L. L. (2008). High-intensity aerobic interval training increases fat and carbohydrate metabolic capacitites in human skeletal muscle. *Applied Physiology, Nutrition, and Metabolism*. Vol 33 pp: 1112-1123.

Perrey. S., and Racinais. S. (2010). Neural and muscular adjustments following repeated running sprints. *European Journal of Applied Physiology*. Vol 109 pp:1027-1036.

Pette. D. (1985). Metabolic heterogeneity of muscle fibres. *Journal of Experimental Biology*. Vol 115 pp: 179-189.

Pette. D. (1998). Training effects on the contractile apparatus. *Acta Physiologica Scandinavica*. Vol 162(3) pp: 367-376.

Pette. D., and Statron. R. S. (1997). Mammalian skeletal muscle fibre type transitions. *International Review of Cytology*. Vol 170 pp: 143-223.

Phillips. S. M., Thompson. R., and Oliver. J. L. (2014). Overestimation of required recovery time during repeated sprint exercise with self-regulated recovery. *Journal of Strength and Conditioning Research*. Vol 28(12) pp: 3385-3392.

Pilegaard. H., Domino. K., Noland. T., Juel. C., Hellsten. Y., Haelstrap. A. P., and Bangsbo. J. (1999). Effect of high-intensity exercise training on lactate/$H^+$ transport capacity in human skeletal muscle. *American Journal of Physiology*. Vol 276 pp: 255-261.

Pincivero. D. M., Lephart. S. M., and Karunakara. R. G. (1997). Effects of rest interval on isokinetic strength and functional performance after short term high intensity training. *British Journal of Sports Medicine*. Vol 31 pp: 229 – 234.

Place. N., Yamada. T., Bruton. J. D., and Westerblad. H. (2010). Muscle fatigue: from observations in humans to underlying mechanisms studied in intact single muscle fibres. *European Journal of Applied Physiology*. Vol 110(1) pp: 1-15.

Pohl. J., Frohnau. G., Kerner. W., and Fehm-Wolfsdorf. Gabriele. (1997). Sympton awareness is affected by the subjects' expectations during insulin-induced hypoglycaemia. *Diabetes Care*. Vol 20(5) pp: 796-802.

Pottgiesser. T., and Schumacher. Y. O. (2013). Current strategies of blood doping detection. *Analytical nd Bioanalytical Chemistry*. Vol 450(30) pp: 9625-9639.

Powers. S. K., and Howley. E. T. (2009). Exercise physiology theory and application to fitness performance. New York, NY, McGraw-Hill.

Proske. U. What is the role of muscle receptors in proprioception? *Muscle & Nerve*. Vol 31(6) pp: 780-787.

Racinais. S., Bishop. D., Denis. R., Lattier. G., Mendez-Villaneuva. A., and Perrey. S. (2007). Muscle deoxygenation and neural drive to the muscle during repeated sprint cycling. *Medicine & Science in Sports & Exercise*. Vol 39(2) pp: 268-342.

Robergs. R. A., Dwyer. D., and Astorino. T. (2010). Recommendations for improved data processing from expired gas analysis indirect calorimetry. *Sports Medicine*. Vol 40(2) pp: 95-111.

Rodas. G., Ventura. J. L., Cadefau. J. A., Cusso. R., Parra. J. (2000). A short training programme for the rapid improvement of both aerobic and anaerobic metabolism. *European Journal of Applied Physiology*. Vol 82 pp: 480-486.

Roepstorff. C., Thiele. M., Hilliq. T., Pilegaard. H., Richter. E. A., Wojtaszewski. J. F. P., and Kiens. B. (2006). Higher skeletal muscle alpha2AMPK activation and lower energy charge and fat oxidation in men than in women during submaximal exercise. *The Journal of Physiology*. Vol 574(1) pp: 125-138.

Romain. M., Phil. W., Hiroshi. H., Bart. S., and Francesca. P. M. (2006). Central Fatigue: The serotonin hypothesis and beyond. *Sports Medicine*. Vol 36(10) pp: 881-909.

Ross. A., and Leveritt. M. (2001). Long-term metabolic and skeletal muscle adaptations to short-sprint training: implications for sprint training and tapering. *Sports Medicine*. Vol 31(15) pp: 1063-1145.

Rossieter. H. B., Kowalchuk. J. M., and Whipp. B. J. (2006). A test to establish maximum $O_2$ uptake despite no plateau in the $O_2$ uptake response to ramp incremental exercise. *Journal of Applied Physiology*. Vol 100(3) pp: 764-770.

Russ. D. W., Lanza. I. R., Rothman. D., and Kent-Braun. J. A. (2005). Sex differences in glycolysis during brief, intense isometric contractions. *Muscle & Nerve*. Vol 32(5) pp: 647-655.

Sahlin. K., Harris., R. C., Nylind. B., and Hultman. E.. (1976). Lactate content and pH in muscle obtained after dynamic exercise. *Pflugers Arch*. Vol 367(2) pp: 143-152.

Schmidt. R. A., and Wrisberg. C. A. (2007). *Motor learning and performance: a situation-based learning approach*. Champaign. Human Kinetics.

Seiler. S., and Hetlelid. K. J. (2005). The impact of rest duration on work intensity and RPE during interval training. *Medicine & Science in Sports & Exercise*. Vol 37(9) pp: 1601-1608.

Seynnes. O. R., de Beor. M., and Narici. M. V. Early skeletal muscle hypertrophy and architectural changes in response to high-intensity resistance training. *Journal of Applied Physiology*. Vol 102 pp: 368-373.

Shepley. B., MacDougall. J. D., Cipriano. N., Sutton. J. R., Tarnopolsky. M. A., and Coates. G. (1992) Physiological effects of tapering in highly trained athletes. *Journal of Applied Physiology*. Vol 72(2) pp: 706-717.

Sloth. M., Sloth. D., Overgaard. K., and Dalgas. U. (2013). Effects of sprint interval training on $VO_2$ max and aerobic exercise performance: A systematic review and meta-analysis. *Scandinavian Journal of Medicine & Science in Sports*. Vol 23 pp: 341–352.

Smith. K. J., and Billaut. F. (2012). Tissue oxygenation in men and women during repeated-sprint exercise. *International Journal of Sports Physiology & Performance*. Vol 7(1) pp: 59-68.

Spriet. L. L., Lindinger. M. I., McKelvie. R. S., Heigenhauswer. G. J., and Jones. N. L. (1989). Muscle glycogenolysis and $H^+$ concentration during maximal intermittent cycling. *Journal of Applied Physiology*. Vol 66 pp: 8-13.

Staron. R. S., Hagerman. F. C., Hikida. R. S., Murray. T. F., Hostler. D. P., Crill. M. T., Ragg. K. E., and Toma. K. (2000). Fiber type composition of the vastus lateralis muscle of young men and women. *The Journal of Histochemistry & Cytochemistry*. Vol 48(5) pp: 623-629.

St Clair Gibson. A., Schabort. E. J., and Noakes. T. D. (2001). Reduced neuromuscular activity and force generation during prolonged cycling. *American Journal of Physiology*. Vol 281(1) pp: 187-196.

Stevens. C. J., and Dascombe. B. J. (2015). The reliability and validity of protocols for the assessment of endurance sports performance: an updated review. *Measurement in Physical Education and Exercise Science*. Vol 19 pp: 177-185.

Swart. J., Lamberts. R. P., Lambert. M. I., St Clair Gibson. A., Lambert. E. V., Skowno. J., and Noakes. T. D. (2009). Exercising with reserve: evidence that the central nervous system regulates prolonged exercise performance. *British Journal of Sports Medicine*. Vol 43(10) pp: 782-790.

Swart. J., Lindsay. T. R., Lambert. M. I., Brown. J. C., and Noakes. T. D. (2012). Perceptual cues in the regulation of exercise performance – physical sensations of exercise and awareness of effort interact as separate cues. *British Journal of Sports Medicine*. Vol 46(1) pp: 42-50.

Tanaka. H., Monahan. K. D., and Seals. D. R. (2001). Age-predicted maximal heart rate revisited. *Journal of the American College of Cardiology*. Vol 37(1) pp: 153-159.

Taylor. C. W., Ingham. S. A., Hunt. J. E., Martin. N. R., Pringle. J. S., and Ferguson. R. A. (2016). Exercise duration-matched interval and continuous sprint cycling induce similar increases in AMPK phosphorylation, PGC-1a and VEGF mRNA expression in trained individuals. *European Journal of Exercise Physiology*. Vol 116(8) pp:1445-1499.

Tucker. R., Marle. R., Lambert. E. V., and Noakes. T. D. (2006). The rate of heat storage mediates an anticipatory reduction in exercise intensity during cycling at a fixed rating of perceived exertion. *Journal of Physiology*. Vol 574 pp: 905-915.

Turner. A. N., and Stewart. P. F. (2013). Repeat Sprint Ability. *Strength & Conditioning Journal*. Vol 35(1) pp: 37-41.

Vandewalle. H., Pérès. G., and Monod. H. (1987). Standard Anaerobic Exercise Tests. *Sports Medicine*. Vol 4 pp: 268-289.

Vanhatalo. A., Doust. J. H., and Burnley. M. (2008). Robustness of a 3-min all-out cycling test to manipulations of power and cadence in humans. *Experimental Physiology*. Vol 93(3) pp: 383-473.

Vigelso. A., Andersen. N. B., and Dela. F. (2014). The relationship between skeletal muscle mitochondrial citrate synthase activity and whole body oxygen update adaptations in response to exercise training. *International Journal of Physiology, Pathophysiology and Pharmacology.* Vol 6(2) pp: 84-101.

Vint. P. F., and Hinrichs. R. N. (1996). Endpoint error in smoothing and differentiating raw kinematic data: An evaluation of four popular methods. *Journal of Biomechanics*. Vol 29(12) pp: 1637-1642.

Warburton. D. E., Gledhill. N., and Quinney. H. A., (2000). Blood volume, aerobic power, and endurance performance: potential ergogenic effect of volume loading. *Clinical Journal of Sport Medicine*. Vol 10(1) pp: 59-66.

Wasserman. K., Whipp. B. J., Koyl. S. N., and Beaver. W. L. (1973). Anaerobic threshold and respiratory gas exchange during exercise. *Journal of Applied Physiology*. Vol 35(2) pp: 236-279.

Weinberg. R. S., and Gould. D. (2006). *Foundations of sport and exercise psychology*. Champaign. Human Kinetics.

Westerblad. H., Allen. D. G., and Lannergren. J. (2002). Muscle fatigue: lactic acid or inorganic phosphate the major cause? *News in Physiological Sciences*. Vol 17 pp: 17-21.

Wit. S., and Dickinson. A. (2009). Associative theories of goal-directed behaviour: a case for animal-human translation models. *Psychological Research*. Vol 73(4) pp: 463-476.

Wittekind. A. L., Micklewright. D., and Beneke. (2011). Teleoanticipation in all-out short-duration cycling. *British Journal of Sports Medicine*. Vol 45(2) pp: 1-6.

Yamagishi. T., and Babraj. J. (2017). Effects of reduced-volume of sprint interval training and the time course of physiological and performance adaptations. *Scandinavian Journal of Medicine & Science in Sports*. doi: 10.1111/sms.12831.

Yamagishi. T., and Babraj. J. (2016). Influence of recovery intensity on oxygen demand and repeated sprint performance. *The Journal of Sports Medicine and Physical Fitness*. Vol 56(10) pp: 1103-1112.

Yu. J., Carlsson. L., and Thornell. L. (2004). Evidence for myofibril remodelling as opposed to myofibril damage in human muscles with DOMS: an ultrastructural and immunoeletron microscopic study. *Histochemistry Cell Biology*. Vol 121 pp: 219-227.

Zając. A., Chalimoniuk. M., Maszczyk. A., Gołaś. A., and Lngfort. J. (2015). Central and peripheral fatigue during resitance exercise – a critical review. *Journal of Human Kinetics*. Vol 30(49) pp: 159-28.